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Minnesota Medicine

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

Volume 29 **February, 1946** **No. 2**

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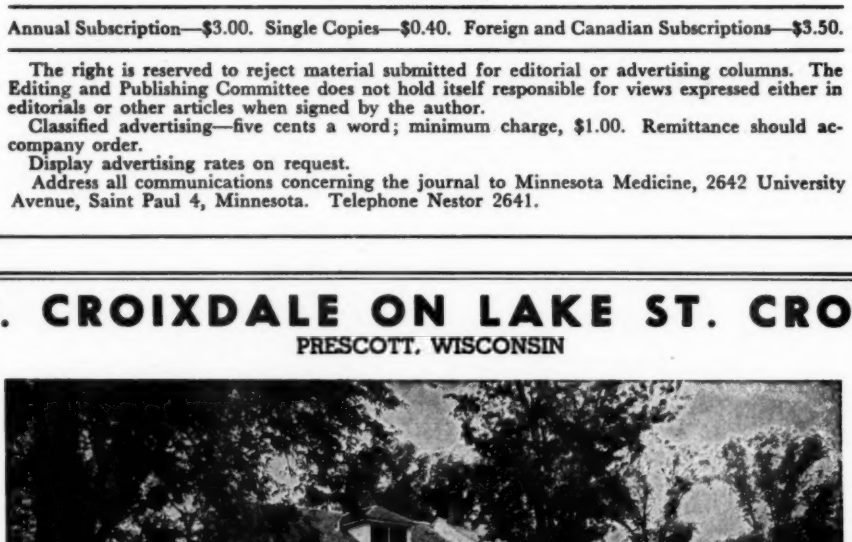

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Prescott, Wisconsin
Tel. 39

Hewitt B. Hannah, M.D.
Joel C. Hultkrans, M.D.
511 Medical Arts Building
Minneapolis, Minnesota
Tel. MAin 4672

SUPERINTENDENT
Ella M. Mackie
Prescott, Wisconsin
Tel. 69

Minnesota Medicine

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

Volume 29 February, 1946 No. 2

THE RH FACTOR IN FEEBLE-MINDEDNESS AND OTHER DISEASES

LAURENCE H. SNYDER

Professor of Medical Genetics and Chairman of the Department of Zoology and Entomology
The Ohio State University, Columbus, Ohio

IN recent years much attention has been devoted to the study of the inheritance of various traits, both normal and abnormal, in human beings. Perhaps the most fascinating of these traits are the recently discovered Rh blood types. At first apparently of only theoretical and academic interest, the Rh factors have become of importance in legal medicine, in transfusions, and in the study of infant mortality, and are now extending their influence into the fields of psychology and psychiatry.

In order to understand the Rh factors and their applications, it is necessary to see them in perspective against the background of the human blood groups in general. And in order to understand the genetic and medical applications of the blood groups, it is in turn desirable to view them in perspective against the background of hereditary human traits and their importance in human welfare. Let me then briefly sketch in broad outline the practical applications of human heredity, adding a little more detail when we come to the blood groups, and finally giving very careful consideration to the Rh blood types.

The educated layman is rapidly learning that much information is now available on the mode of inheritance of specific traits in human beings, and he is responding to this awareness by asking questions of his physician, his visiting nurse, his pastor, and other social workers. It behooves those involved in social service to learn some of the answers, and to know where to obtain further information which they themselves may not possess. You in Minnesota are to be congratulated for having available the Charles Fremont Dight Institute for the Study of Human Genetics, which is serving admirably as a clearing house for such information, as well as in a research capacity.

One of the important practical applications of our increasing knowledge of human inheritance is our ability to give genetic advice to families. Parents and prospective parents are often concerned about the future appearance in their children of a trait which has previously occurred in the family. Sometimes it is a desirable trait such as musical ability, curly hair or high intelligence. Sometimes it is an unwanted trait such as diabetes, cleft palate or feeble-mindedness.

Before attempting to answer such a question the consultant must take several steps. He must first make sure of the accurate identification of the trait. This frequently means a medical diagnosis. The ease of identification or diagnosis varies: cleft palate is recognized with relative ease, borderline mental deficiency with considerable difficulty. Frequently, minor variations within a trait must be carefully noted, as they may represent genetic entities.

Having properly described and identified the trait, the second step is to chart the family history of the person seeking advice. This is essential in making an accurate prognosis. The third step is to compare this pedigree with those already re-

A public lecture delivered at the University of Minnesota under the auspices of the Charles Fremont Dight Institute and the Minnesota Human Genetics League, November 14, 1945.

corded in the literature or in the files of the consultant, noting the type of hereditary behavior previously assigned to the trait, and paying careful attention to variations in the expression of the trait, the age of onset, and the regularity with which the character has appeared.

Last, the potential effects of the environment on the trait must be reviewed, and the probable environment in which the trait will develop if it appears in this family must be specified.

With the foregoing information available it may be possible to give a reasonable estimate of the probability of the recurrence of the trait.

A few personal instances may be permissible as illustrations. I was recently consulted by a man who had suffered all his life from brittleness of bones, fractures occurring at the slightest provocation. The sclerotic coat of the eyeball was pale blue. His mother had had the same blue sclerotics, but with less marked tendency to fractures. He wished to marry, and wondered whether the trait might reappear. Since it is known to be transmitted as a dominant factor, it was necessary to inform him that he might expect the condition in about half his children, both boys and girls.

I was asked to advise a young couple who had lost their first child from the infantile form of amaurotic idiocy. This is a condition in which mental failure, paralysis, blindness and finally death occur in infancy. The diagnosis had been made by a competent physician. The mode of inheritance of this condition is known to be that of an autosomal recessive factor, unaffected by ordinary environmental influences, so that the parents were notified that the chances of another child having the disease were one in four. In this instance the parents felt that they were emotionally unable to stand another strain like that imposed by the slow death of the first child, and decided to adopt a child.

Very recently there appeared at the Ohio State University Hospital two brothers, aged nine and eleven, suffering from pseudohypertrophic muscular dystrophy. The parents had come to appreciate the hopeless outlook for the affected boys, but wanted particularly to know the chances that the condition would reappear if the normal brothers and sisters should marry. Since the dystrophy is the result of a sex-linked recessive factor, it was possible to advise them that the normal boys in the family could not possibly transmit the defective gene, but that the girls had an even chance of carrying it and thus of producing affected sons.

A short time ago I was asked to see a young couple who had a male infant suffering from a condition known as epidermolysis bullosa of the dystrophic type. In this form of the abnormality the skin becomes blistered wherever subjected to friction, the blisters occurring between the epidermis and the corium. They become malignant, and death usually occurs in infancy or childhood. The nails and teeth are affected, and the blisters occur in the mucous membranes as well as in the more usual places. The parents wanted information as to future children. This type of epidermolysis is due to an incompletely sex-linked gene, so that a still different prognosis must be made. Since the affected child was a boy, other affected children in this family are likely to be boys, although occasionally an affected girl might occur. With a known crossover value of twenty per cent for this trait, a girl in this family would have only one chance in ten of being affected, while another boy would have nearly one chance in two (four in ten) of having the epidermolysis.

The foregoing instances are all examples where sufficient information on the genetics of the trait was available to make accurate prognosis possible. In many diseases and abnormalities, accurate genetic data are not at hand and explicit information cannot be given. There is crying need for more and more research in medical genetics and human heredity.

One of the most important practical applications of our knowledge of human genetics is in the field of preventive medicine. It is becoming more and more feasible to prevent, in the relatives of persons with genetic conditions, the appearance of the abnormality or disease. It is done by instituting preventive measures, on the basis of the genetic history. The procedure is to examine the relatives of an affected patient for the early pre-clinical or laboratory signs of the trait, and then to take such precautions in those who show the signs as will minimize the danger. The preventive splenectomy in hemolytic icterus is a brilliant example of such procedures, and many others could be cited.

To take a personal instance, we were recently able to suggest such preventive measures in a family in which several members suffered from xanthoma tuberosum. This, like the amaurotic

idiocy previously referred to, is a storage disease, a lipoidosis. In contrast to amaurotic idiocy, however, there are pre-clinical laboratory signs which may be discovered in time to prevent any fatal consequences. The visible signs of the abnormality are nodules and tumors of the joints and tendons. The hidden symptoms are increases in the blood cholesterol and cholesterol esters. In contrast to a normal blood cholesterol of 150-300 mg. per cent, the value in xanthoma may reach 1500 mg. per cent. The disease may progress to cardiovascular involvement with sudden death.

Since the increase in blood cholesterol is transmitted as a dominant factor, and since it may occur long before the appearance of the nodules and tumors, it is feasible to examine the relatives of patients with nodules by determining their blood cholesterol and cholesterol esters. Where hypercholesteremia is found, preventive dietary measures may be instituted, thus helping to preclude cardiovascular involvement and possible death.

Another important set of practical applications of human genetics occurs in medico-legal cases as a consequence of our knowledge of the human blood groups. In the early days of this century Karl Landsteiner discovered that when the red cells of one person were mixed with the serum of another person, a reaction sometimes occurred. The red cells were clumped, or agglutinated, by the serum. This did not happen in all mixtures of serums and cells, but only when the cells of certain persons were mixed with the serum of certain other persons. Landsteiner recognized that the reaction was that of antigen with antibody; that is, the red cells of some people contain an antigen, and the serum of some others contain the corresponding antibody. Upon mixture of the antigen and the antibody, agglutination results.

Further work by Landsteiner disclosed the fact that there were two such antigens in human blood, and these he named A and B. There are thus four kinds of people in the world: those containing antigen A in their cells (hence spoken of as belonging to group A), those containing antigen B (group B), those having both antigens (group AB), and those having neither antigen (group O).

There is a reciprocal relationship between the antigens and the antibodies in the A-B series, since everyone has either antigen A or the antibody against A, and everyone has either antigen B or the antibody against B. There are thus natural or "normal" antibodies against A and B available in the bloods of various individuals.

TABLE I. MAJOR LAWS PERTAINING TO MEDICO-LEGAL APPLICATIONS OF BLOOD GROUPS AND TYPES

1. Agglutinogen A, B, M, N, P, Rh or Hr cannot appear in a child unless it was present in one or both parents.
2. Water-soluble agglutinogen A or B (as opposed to alcohol soluble) cannot appear in a child unless one or both parents had the "secreting" ability.
3. A parent of group AB cannot give rise to a child of group O.
4. A parent of type O cannot give rise to a child of group AB.
5. A parent of type M cannot give rise to a child of type N.
6. A parent of type N cannot give rise to a child of type M.
7. A parent lacking a particular Rh agglutinogen cannot give rise to a child lacking the corresponding Hr agglutinogen.
8. A parent lacking a particular Hr agglutinogen cannot give rise to a child lacking the correspondeing Rh agglutinogen.

The first obvious importance of this discovery was to transfusion, since it would not do to give red cells to a person whose serum would immediately clump these cells. Hence in all transfusions the blood groups of donor and recipient must be determined, and compatible blood given.

The fact that there were four kinds of people who could be classified in this way was of immediate interest to the geneticist. In our own laboratory and those of other investigators researches were quickly undertaken to determine how the blood groups were inherited. The study of many thousands of families has made it clear that antigen A never appears in a child's blood unless it was in the blood of at least one of the parents. Similarly, antigen B never appears in a child's blood unless it was present in the blood of at least one of the parents. Other more complicated laws can also be stated, which need not be gone into in detail here (Table I).

Although these facts were at first merely of academic interest, they proved very quickly to be of practical importance. They may be used, for example, in cases of disputed parents. If a child, for instance, is of group B, and the mother is of group O, then we know that the child inherited antigen B from its father. If the alleged father should be of group O or of group A, we can say definitely that he is not the true father of the child.

Antigen A was later found to occur in three grades, so to speak. These have been namd A_1, A_2 and A_3. There are thus actually eight different groups in the A-B series, namely O, A_1, A_2, A_3, B, A_1B, A_2B, and A_3B.

You will recall that antigens A and B were discovered because of the presence in human serums of naturally occurring antibodies against them. In 1927 it occurred to Landsteiner and Le-

vine that there might be still other antigens present in blood cells for which there were no normal antibodies. Such antigens might be discovered by injecting human red cells into animals, in which case the animals would produce antibodies against any specific antigens present in the cells. Accordingly they injected group O cells into rabbits, removed the heterologous antibodies, and discovered that the rabbits had produced specific antibodies against several antigens not previously recognized in human blood cells.

Two of these newly discovered antigens were named M and N. They were found to be related in such a way that a person might have one of them in his cells, or the other, or both. No one has been found who lacks both M and N. In inheritance studies, it was found in our laboratory and others that these antigens, like A and B, are dominant, and do not appear in a child's blood unless they were present in the blood of at least one of the parents. Like antigen A, antigen N comes in more than one grade. Thus N_1 and N_2 are recognized, and there are five types in the M-N series, as follows: M, N_1, N_2, MN_1, MN_2. Within the A-B series, it will be recalled, there were eight kinds. Since any one of those eight could be any one of the five M-N types, there are 8 x 5, or 40 different groups in these two series taken together.

Not only do antigens M and N lack normal antibodies, but they are only very weakly antigenic to human beings. Hence they are of no importance in transfusions, since even multiple injections of M into a person lacking it, or of N into a person lacking it, will not produce strong antibodies, such are produced by injection into rabbits. The chief importance of antigens M and N is in their medicolegal applications, in disputed paternity cases (Table I).

Another antigen was found in the course of the foregoing studies of Landsteiner and Levine. This antigen was named P, and is inherited independently of antigens M and N. It is less satisfactory to use in medicolegal studies than the other antigens, since potent sera are difficult to obtain and standardize, and the reactions are graded. Certain normal animal sera have been found to be fairly good sources of anti-P, particularly pig serum. Like the previously mentioned antigens, P does not occur in a child's blood unless it was present in the blood of at least one of the parents. Moreover, P also appears to occur in two grades, P_1 and P_2, so that four kinds of persons can be classified when proper antisera are available, namely, P_1, P_2, P_1P_2, and P—.

It will be recalled that in the combined A-B and M-N series there were 40 different types of human blood. Since any one of the 40 could be any one of the 4 types in the P series, there are altogether 40 x 4, or 160 types, using all the antigens so far discussed.

In the years between 1930 and 1933 various investigators demonstrated that antigens A and B occur in two main chemical forms: alcohol-soluble and water-soluble. Thus persons of groups A, B, and AB may develop water-soluble antigens, in which case the group-specific substance is found in the body fluids (saliva, tears, urine, milk, etc.) as well as in the cells. Or they may develop alcohol-soluble antigens, in which case the group-specific substance is restricted to the blood cells and tissue cells. Schiff and his coworkers have designated those persons having water-soluble antigens as "secretors," those having alcohol-soluble antigens as "non-secretors." By means of special techniques even individuals of group 0 can be classified into secretors and non-secretors.

The water-solubility of antigens A and B, that is to say, the "secreting" ability, is inherited on the basis of a dominant factor. When both parents are non-secretors, all the children are non-secretors. These factors may thus be added to these previously used in medicolegal applications (Table I). In addition, they double the number of recognizable blood types. Using antigens A, B, M, N and P, we have already classified 160 types. Since any one of these 160 types could be S+ (secretor) or S— (non-secretor), there would be 160 x 2, or 320 types thus far classifiable.

We have now set the stage for the discussion of the Rh blood types, which fit into the picture at this point.

Further attempts at discovering still more human blood antigens by injection of human blood into animals gave disappointing results. Landsteiner and Wiener therefore tried a somewhat different approach. In 1940 they injected blood from the monkey, *Macacus rhesus*, into rabbits. The rabbits responded by producing an antibody against rhesus blood. When this antibody was tested against human blood, it was found to agglutinate the cells of about 85 per cent of white individuals. Thus 85 per cent of us share an anti-

gen in common with rhesus monkeys, and 15 per cent of us do not. This antigen is different from A, B, M, N or P. Using the first two letters of "rhesus," the antigen was named Rh. Persons having the new antigen were designated Rh positive (Rh+), while those not having it were designated Rh negative (Rh—).

studied the blood of a mother who harbored a macerated fetus, and found that it contained an atypical antibody. They postulated that the mother had been immunized by her own fetus, and the resulting antibody had caused the destruction of the fetus. The following year Landsteiner and Wiener announced the discovery of the Rh factor,

TABLE II. HUMAN BLOOD GROUPS

System	Groups	Classes
A-B-O	O, A_1 A_2, A_3, B, A_1B, A_2B, A_3B	8
MN	M, N_1, N_2, MN_1, MN_2	5
P	P_1, P_2, P_1P_2, P–	4
S	S+, S–	2
Rh	Rh°, Rh°′, Rh°″, Rh°′Rh°″, Rh′, Rh″, Rh′Rh″, Rh–	8

8 × 5 × 4 × 2 × 8 = 2,560

Studies on the inheritance of the Rh antigen showed that it too was inherited as a dominant factor, and that it never appeared in a child's blood unless it was present in the blood of at least one of the parents. It therefore takes its place with the other antigens in medico-legal applications (Table I).

There are no normal antibodies against antigen Rh in human serums. In this respect the situation is similar to that in regard to M and N. Unlike antigens M and N, however, Rh is highly antigenic to human beings. Repeated injection of Rh+ blood into an Rh— person results in immunization of the recipient, and transfusion reactions of increasing severity. It is essential, therefore, to transfuse Rh— blood into an Rh— person. For even though the first transfusion might cause no reaction, the immunization process would be initiated, and a subsequent transfusion might be fatal. While only a small proportion of transfusions in civilian practice is made up of multiple transfusions, this proportion rises in military medicine to 80 per cent.

The Rh antigen has another unique property. It is capable of passing from the fetal circulation through the placenta into the maternal circulation. This fact led to another important discovery, namely, the cause of the disease known as erythroblastosis.

Erythroblastosis is a disease of the fetus or newborn infant, characterized by anemia, jaundice, congenital hydrops, and sometimes stillborn macerated fetuses. In 1939 Levine and Stetson and this quickly proved to be the agent postulated by Levine and Stetson.

Levine, Katzin and Burnham in 1941 showed that the Rh antigen was indeed the primary cause of erythroblastosis fetalis. In nearly all cases of the disease, the mother proved to be Rh—, the fetus Rh+. The Rh antigen in the fetus was, of course, inherited from the father. The Rh— mothers can be shown to have developed antibodies against Rh. Apparently the Rh antigen has passed through the placenta into the circulation of the mother, immunized the mother, and the maternal immune antibody has then passed back into the fetus, damaging the red blood cells. The result is hemolytic jaundice and other effects. While the test-tube reaction is agglutination, the end result in the fetal circulation is hemolysis. The regenerative activity of the fetal blood-forming system floods the circulation with immature red cells (erythroblasts), hence the name erythroblastosis.

Ordinarily the first Rh+ child born to an Rh— mother is itself unaffected, since the production of antibodies by the mother has not reached a high titer by the time of the birth of the infant. A second Rh+ embryo, however, stimulates the further rapid production of antibodies, and this and all subsequent Rh+ fetuses will be affected. Occasionally a woman produces antibodies so readily that even the first Rh+ embryo may be affected. Of course if an Rh— woman has received a transfusion of Rh+ blood previous to her first pregnancy, she will already have been immunized,

and the first Rh+ fetus she carries can be affected by the antibodies.

On the other hand, some women produce antibodies so slowly that several Rh+ fetuses may be required before one becomes erythroblastotic.

Although anti-Rh serum was originally obtained from rabbits immunized with rhesus monkey blood, and later from immunized guinea pigs, it was soon found that the serum from mothers of erythroblastotic infants was a much better source of test antibodies than serum from immunized animals. Such mothers are the present source of test serums. In studying human serums from immunized mothers, several new kinds of anti-Rh antibodies have been discovered, making it possible to identify corresponding new kinds of Rh antigens.

Shortly after the description of the original Rh antibody, Wiener found a human serum which agglutinated only 70 per cent of human bloods, but which was related to the original anti-Rh serum, since bloods negative to the one were usually negative to the other. The original antigen is now called Rh° (or Rh_o), and the new one is called Rh'. Levine then found a serum which agglutinated about 87 per cent of human bloods, and obviously contained both antibodies, that against Rh° and that against Rh'. Some persons are found to have Rh° in their red cells, and some to have Rh'. In addition, some persons have both, and this may be the result of their inheriting two genetic factors, one for each antigen, or of their inheriting a single genetic factor (*Rh°'*) accounting for both.

Still later Wiener discovered a third type of human antiserum which reacted with but 32 per cent of human bloods. The antigen corresponding to this antibody was named Rh". Using the three antiserums, eight kinds of people may be distinguished, as follows: Rh°, Rh', Rh", Rh°', Rh°", Rh'Rh", Rh°' Rh°", and Rh—. The first seven of these eight types are Rh+ for one or more of the Rh antigens; the last one is Rh negative.

We had already distinguished 320 kinds of people based on A, B, M, N, P and S. Since any one of the 320 could be any one of the eight Rh types, we can now distinguish 320x8, or 2,560 types, using all the known factors (Table II). Of course some of the types are far commoner than others in the population, and some of the antiserums are difficult to obtain in standard form. Nevertheless, with the proper antiserums it would be possible to take a small sample of blood from each one of you in the audience, and in a short time to place each of you in your own blood type. It would be surprising if any two of you were in the same type. Ten years from now it would be possible to take another sample of blood from each of you, and without having the samples individually named, to assign each blood to a specific person by referring to the previously recorded data, except for such duplications of type as might exist among you.

Using all the Rh factors, 87 per cent of American whites are Rh+ and only 13 per cent are Rh—. Taking these figures, we may compute how often Rh— women will marry Rh+ men by chance in the general population, and how often such marriages should result in Rh+ children, who might thus have erythroblastosis. Let us for this purpose consider all the Rh antigens collectively, as Rh+, contrasted with Rh—, in which none of the antigens is present. Since hereditary factors occur in pairs in an individual, an Rh+ man might be *RhRh* or *Rhrh*. In the first instance he is said to be pure or homozygous for the Rh factor, and can transmit only the factor for the antigen to his children. In the second instance (*Rhrh*), he is hybrid, or heterozygous, and will transmit the factor for the Rh antigen to only half his children. Rh— women are all homozygous *rhrh*.

By appropriate statistical methods of gene-frequency analysis, we can show that in the general population 41 per cent are *RhRh*, 46 per cent are *Rhrh*, and 13 per cent are *rhrh*. Thus Rh— women will marry Rh+ men in 11.3 per cent of all matings. But 5.33 per cent (13 per cent of 41 per cent) will be with homozygous men, and 5.98 per cent (13 per cent of 46 per cent) will be with heterozygous men. All the offspring from homozygous men will be Rh+, but only half of those from heterozygous men will be Rh+. Therefore 5.33 per cent + ½ of 5.98 per cent of all children born will be Rh+ children from Rh— mothers. This figure is 8.32 per cent. We should expect to find erythroblastosis, then, in 8.32 per cent of all births, that is, about one in twelve. However, the frequency of clinically diagnosed cases has never approached this figure. The recorded incidence is about one half of one per cent; or about one in 200 births. Why does this discrepancy between the calculated and the observed proportions occur?

First of all it must be remembered that first-

born children are seldom if ever affected. In the American population, the average number of children in families having at least one child is 3.24. This means that one out of 3.24, or about 31 per cent of children born in this country, are firstborn. This reduces the expected proportion of children showing the effects of the immunization of the mother by the Rh factor to 5.74 per cent. Thus we would expect only about one in seventeen to show the effects. This closes somewhat the gap between expected (1 in 17) and observed (1 in 200), but still leaves a considerable discrepancy.

The cases of erythroblastosis are not distributed at random among the potential Rh— mothers, but usually occur several in the family. It may be, then, that only some women are capable of producing potent antibodies against the Rh antigens. It may also be true that the antigen gets through the placenta only in some Rh— mothers. Perhaps both of these phenomena account for some of the discrepancy between calculated and observed incidences of erythroblastosis.

It is also conceivable, however, that in some instances where the antigen penetrates the placental barrier, and the antibodies in turn reach the fetal circulation, that the effects on the fetus are different from those recognized as classical erythroblastosis.

In various laboratories, including our own, the search has been made for such manifestations. The effects of Rh immunization appear to be unimportant in the production of early abortions, and in the etiology of hemolytic icterus, sickle cell anemia, hydatiform mole, ectopic pregnancies, eclampsia and specific toxemia.

In 1944, Yannett and Lieberman made an examination of the blood groups and types of the children in a school for the feeble-minded, and of their mothers. There were too many Rh— mothers in the group, and too many Rh+ children of Rh— mothers. Breaking down the results it was found that of 109 feeble-minded children, fifty-three were distributed among the specific types of mental deficiency such as Mongolian idiocy and spastic paraplegia. The distribution of the Rh factor was normal in these children and in their mothers.

In the fifty-six children classified as undifferentiated mental deficiency, however, an abnormal distribution of the Rh factor was found. There were too many Rh— mothers and too many Rh+ children from these Rh— mothers.

These results appeared to us so suggestive that we have been investigating the blood of the undifferentiated feeble-minded at the Ohio Institution for the Feeble-minded, and of their mothers (Snyder, Schonfield and Offerman, 1945). The complete results up to date are as follows: Of 169 mothers of feeble-minded children, thirty-eight are Rh—, whereas only twenty-one or twenty-two would be expected. This difference is statistically highly significant. Of 171 feeble-minded children, twenty-seven are Rh+ from Rh— mothers, whereas we should expect only fourteen. This deviation is also highly significant when tested statistically.

Thus it would seem that in some instances the immune antibodies of the mother produce effects on the brain tissue instead of the usual effects recognized as erythroblastosis. It has been suggested that the immediate effect of red-cell destruction is anoxia, and that this lack of oxygen, if it occurs at a time when the brain of the embryo is in a critical stage of development, may very well cause permanent mental deficiency.

Thus the gap between the calculated incidence of effects of Rh immunization and the observed incidence has been partially closed up. However, the search must continue for further, as yet unknown, manifestations. In our laboratory we have recently completed an analysis of a large number of cases of dementia praecox and their mothers. The distribution of the Rh factors is perfectly normal in these instances.

It might seem surprising that heterospecific pregnancies in regard to antigens A and B play no part in erythroblastosis, since antibodies against A and B are normally present in bloods lacking these antigens, and would not even have to be stimulated by immunization. The explanation seems to be that antigens A and B, at least in secretors, are found in all tissue cells and body fluids, including the amniotic fluid and secretions of the fetus. This may serve to protect the fetus from antibodies against A and B. On the other hand, antigen Rh seems to be restricted to the red cells, and such a protective mechanism cannot operate. There is, however, some evidence that incompatibility of A and B can cause mild symptoms in cases where the fetus has one of these antigens which is lacking in the mother. Mild

TABLE III. GENETIC BASIS OF THE RH BLOOD TYPES, SHOWING REACTION OF EACH TO RH AND HR ANTISERA.
(Frequencies given are for American Whites)

Types	Frequency	Genotypes	Anti Rh° .85	Anti Rh′ .70	Anti Rh″ .32	Anti Hr° .63	Anti Hr′ .80	Anti Hr″ .97
Rh°	.02	Rh°Rh° Rh°rh	+ +	— —	— —	— +	+ +	+ +
Rh°′	.52	Rh°′Rh°′ Rh°Rh′ Rh°′Rh° Rh°′rh Rh°′Rh′	+ + + + +	+ + + + +	— — — — —	— + — + +	— — + + +	+ + + + +
Rh°″	.16	Rh°″Rh°″ Rh°″Rh″ Rh°″Rh° Rh°″rh Rh°Rh″	+ + + + +	— — — — —	+ + + + +	— + — + +	+ + + + +	— — + + +
Rh°′Rh°″	.15	Rh°′Rh°″ Rh°′Rh″ Rh°″Rh′	+ + +	+ + +	+ + +	— + +	+ + +	+ + +
Rh′	.015	Rh′Rh′ Rh′rh	— —	+ +	— —	+ +	— +	+ +
Rh″	.005	Rh″Rh″ Rh″rh	— —	— —	+ +	+ +	+ +	— +
Rh′Rh″	.—	Rh′Rh″	—	+	+	+	+	+
Rh–	.13	rhrh	—	—	—	+	+	+

Rh° also called standard Rh
Rh°′ also called Rh$_1$
Rh°″ also called Rh$_2$

jaundice of group A infants from group O mothers has been reported by Polayes (1944). In these cases the titer of anti-A antibodies was found to be 1:700 as compared with an average of less than 1:200.

At first glance it might appear that the death of erythroblastotic infants is gradually eliminating the *Rh* factor. Actually it is the other way around, and the *rh* factor is being eliminated instead. This is due to the fact that every erythroblastotic infant is heterozygous, *Rhrh*, so that each death removes equal numbers of the two genes. Since there are far more *Rh* genes than *rh* genes in the world, a larger proportion of the *rh* genes is lost each time.

The mechanism of the immunization of the mother has been discussed by various workers (cf. Levine 1944). It is assumed that in every normal pregnancy minute quantities of blood from the fetus may find their way into the maternal circulation through microscopic breaks in the villi of the placenta. Very small amounts of blood are necessary for immunization. When it is realized that the total area of villi exposed in the placenta to the maternal circulation is 70 square feet, and that these villi, if extended in a straight line, would reach eleven miles (Dodds 1922), it is not hard to imagine the availability of microscopic amounts of fetal blood to the mother.

The feeble-mindedness due to Rh incompatibility is an acquired mental deficiency, and cannot of course be transmitted to offspring. It could thus account for some of the isolated cases of undifferentiated feeble-mindedness in otherwise highly intelligent families, and also for some of the instances where two feeble-minded parents have produced normal offspring. Perhaps not more than 10 per cent of all undifferentiated mental deficiency can be attributed to Rh incompatibility. The mere fact that it occurs, however, means that when an Rh— girl marries an Rh+ man, she is exposing herself not only to the risk of stillborn or erythroblastotic children, but of mentally deficient children as well.

While these facts are not cause for undue alarm, it would seem advisable that every girl, on entering into marriage, should learn her Rh type and that of her husband. If she is Rh+ (as seven out of eight will be) she has nothing to worry about. If she is Rh— and marries an Rh— man, she still has no cause for worry. But if she is Rh— and marries an Rh+ man, she should in-

form her family physician of this fact, so that he may take all available precautions for the preservation of any Rh+ infant she might have.

Recent discoveries have brought both new dangers and new hopes. I refer to the newly described Hr factors (Hr is Rh in reverse). Once in a great while an Rh+ mother has an erythroblastic child. The danger is very small indeed, but the hopes aroused by this discovery are great. In these cases the effect is that of the Hr factor. The Hr factor is reciprocally related to the Rh factor. Corresponding to the three Rh antigens, Rh°, Rh′ and Rh″, three Hr antigens, Hr°, Hr′ and Hr″ are postulated. Two of these, Hr′ and Hr″ have been discovered and described; the third will no doubt soon be found. The reactions of the blood types with Rh and Hr antiserums are shown in Table III.

The rare cases of an erythroblastotic child born to an Rh+ mother are cases where the mother is Hr— and the child is Hr+. The Hr— mother and the Hr+ child must both be Rh+, since as shown by the table an Hr— mother cannot be heterozygous for *rh*. The father may be of any Rh type, since Hr+ individuals occur in all Rh blood types.

The hopes aroused by this new discovery are that by the use of anti-Hr serums we may be able to pick out, in some instances at least, heterozygous Rh+ men. This would be of value because such men, when married to Rh— women, could expect half their children to be Rh— and therefore free from effects of the incompatibility, whereas homozygous Rh+ men cannot produce Rh— offspring.

Using antigen Hr′, there are ten kinds of Rh types instead of eight, as may be seen from Table III. There would thus be 3,200 different types classifiable instead of 2,560. Using all three Hr antigens when they become available, we shall have eighteen Rh types, and thus 5,760 blood types altogether. Looking into the future, the final result staggers the imagination. The specificity of human blood is a fascinating subject for research.

The Hr antigens are very new to us as yet, and are at present only partially understood. It is obvious that much detailed research work remains to be done on this and other phases of the problem. Basic research in chemistry, physics, biology and medicine will all contribute to its solution. The problems involved are primarily problems of medical genetics, however, and can be solved only by those trained in medicine and in genetics, working in co-operation. May I take this opportunity to point out the very valuable potentialities of integrated groups like those working at the Dight Institute, and may I urge you all to give your fullest support to such programs of research and education as they are carrying out.

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MASS X-RAY SURVEYS

The results of mass x-ray surveys in discovering unsuspected tuberculosis in apparently healthy persons lead one to the logical assumption that just as much, and probably more, tuberculosis might be found through routine roentgenological examination of patients admitted to our general hospitals and clinics. The Mayo Clinic has had such a program for a number of years. At the University of Chicago Hospital and Clinics where this procedure has been followed since 1939, 1.43 per cent of those examined have shown clinically significant tuberculosis. In addition, a large number of nontuberculous chest conditions were found. The most important among these were malignant neoplasms and cardiovascular disease. The over-all percentage of pathological findings was 21.2 per cent.—KARL H. PFUETZE, M.D., Med. Dir. & Supt., Mineral Springs San., Cannon Falls, Minn.

SEROLOGY AND OBSTETRICS

R. T. La VAKE, M.D.
Minneapolis, Minnesota

THE purpose of this paper is to discuss the accumulating obstetrical serologic evidence that suggests: that the definition of the A, B, and Rh antigens as merely agglutinogens is inadequate; that in humans their essential property is that of toxins when they can gain access to a host that does not possess them by inheritance; that in the manifestations of pregnancy toxemia we are witnessing the effects in the host of one or more of these toxins, whereas in erythroblastosis and some fetal deaths and abortions, we are witnessing the effects of corresponding specific antitoxins, generated or inherited by the host.

Though an obstetrical topic, the practical bearing of the evidence on the problems of safe blood transfusion is of importance to all.

Clinical and pathological data have increasingly converged in proof upon the fetal toxin hypothesis of pregnancy toxemia. Data have been furnished by Veit, in 1902; Dienst, in 1905; James Young, in 1914; McQuarrie, in 1923; Ottenberg; and Bartholomew and Colvin; to name but a few investigators. These data, though raising the probability of the fetal toxin hypothesis was not conclusive evidence. To be conclusive, a clearly identifiable toxic substance or antigen must be demonstrated in the fetus, and a corresponding antitoxin or antibody in the mother, and a functional relationship between them must be demonstrable, precisely as obtains in bacterial toxemia or snake venom poisoning.

The serological approach to the pregnancy toxemia problem was unsatisfactory until Jonsson, in 1936, showed that by titering the maternal group antibodies one could often predict the group of the child by the rise above average of the titer of the maternal antibody involved. For example, in a group O mother, with a group B husband, if the B antibody titer rises above average, it allows one to predict with fair accuracy the B status of the child. It has been in such cases where the maternal antibody titer rises that the obstetrician has been finding the manifestations of toxemia. One logical interpretation is that the rise of the titer is a manifestation of an antitoxin response to its corresponding toxin. This seemed like valid reasoning, but how could one account for the cases of toxemia in which no A or B incompatibility existed between child and mother? This had been an insistent question since the beginning of this work in 1919. Possibly the toxin was more fundamental than could be demonstrated by blood group incompatibility. Possibly men were correct in maintaining that the A and B substances were merely agglutinogens and non-toxic antigens. To throw some additional light on the problem, a maternal skin test was devised for toxemics, which, like the Mantoux test for tuberculosis, was interpreted as showing that whatever the nature of the toxin, a toxin existed, and in pregnancy toxemia the mother was immunized to that particular toxin. It seemed to aid in the proof of the toxin hypothesis, but it in no way identified the toxin and could only be used after the birth of the child.

Then came the discovery of the so-called Rh factor by Landsteiner and Wiener, followed shortly by the work of Levine, Katzin and Burnham, showing the relationship between this factor and erythroblastosis fetalis, abortions, stillbirths and macerated fetuses. To the investigator of the toxemias of pregnancy, these discoveries took on a much wider significance than was manifest in their obvious importance in blood transfusion and erythroblastosis. The discovery of the Rh factor furnished a possible answer to the insistent question above referred to, and the work on erythroblastosis furnished further evidence in the fetal toxin argument by completing the missing arc of the circle of the classical and accepted toxin-antitoxin mechanism. If the incompatible fetus, which is a colony of genetically regimented cells, each containing a toxin specific to that mother, could suffer injury and death from the mother's specific antitoxin, then it would be unreasonable to support a belief that the maternal organism could emerge without showing signs of injury. The maternal manifestations must be ascribed to the action of the

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toxin, as in bacterial or snake venom poisoning. At first, the Rh factor held the field, but it soon became apparent that the A and B antigens could bring about erythroblastotic injury and disaster where the Rh status of the husband and wife were similar. All this data irresistibly leads the obstetrician to the conclusion that the only adequate definition of the A, B, and Rh antigens must be that they are toxins. That is their essential property. The relationship of the fetus to its mother is a toxin-antitoxin relationship, when the fetus possesses one or more of these toxins and the mother does not. The results depend upon whether these toxins can gain access to the mother, the amount of the insult, and the reactive properties of the maternal organism.

At first it was perplexing to note that only about 30 per cent of the erythroblastosis cases showed gross signs of pregnancy toxemia. If only 30 per cent showed signs of toxemia and 70 per cent did not, perhaps the men were correct who ascribed all the manifestations of toxemia to the cardio-vascular-renal setup. The advocates of the fetal toxin hypothesis had always held that the cardio-vascular-renal setup played the role of the soil upon which the toxic pregnancy was implanted. It functioned as a conditioning agent but not as a cause. Were we overlooking subclinical toxemia in the missing 70 per cent? It suggested itself that the same law might function here as in snake venom poisoning, namely, that, all things being equal, the manifestations of poisoning in the host vary directly with the strength and amount of the toxin, and indirectly with the strength and amount of the antitoxin generated in the host, or given artificially. A study of maternal antibody titers in the past five years is highly suggestive of the similar functioning of this very law. If one will follow the specific antibody involved in any toxemia, one will see that when the titer remains comparatively low, the manifestations of toxemia are the most severe. After delivery, upon separation of the fetus with its antibody absorptive power, the maternal antibody titer will jump to many times its antepartum strength, with a rapid subsidence of the toxic manifestations. The highest jumps occur in cases of erythroblastosis in which the mother has shown no evidence of toxemia. The interpretation here is that the antibody or antitoxin absorptive power of the fetus keeps the maternal antibody titer comparatively low, but high enough to protect the mother and yet it is high enough to injure or kill the fetus.

Antibody studies suggest that a toxin-antitoxin basis forms the link between late toxemia and premature separation of the placenta. This basis furnishes the only reasonable explanation for certain types of onset and for some of the bizarre findings at times exhibited where for any reason the abdomen has been opened in cases of toxemia or premature separation. One of the interesting features of this serologic approach is that you can check back for years over your outstanding cases if the mothers and children are available.

The following of antibody titers also brings out another suggestive finding. A titer will run along on a virtual level, when, with the accession of an acute infection, the titer will jump from ten to twenty times its former strength, remain there during the infection, and then return to its preinfection level. This finding furnishes one rational explanation for the 40 to 50 per cent correlation between severe infection and death of the fetus and abortion. Although fetal red cells, as shown by Kemp, are five times less vulnerable to antibody agglutination than are adult cells (and one should thus be able to predicate logically that all the fetal cells are in this proportion protected from the deleterious effects of any antibody) if, by infection, any antibody strength were disproportionately raised, the possible results to the fetus would be obvious. Clinical observation suggests that not alone by direct action is the fetus killed by the maternal antitoxin, but indirectly through placental infarction. The placenta is the first line of defense in the toxin-antitoxin battle, and it would appear that infarction is a consequence of toxin-antitoxin action. This would explain the high correlation between the finding of gross infarction and late toxemia, and, in relation to infection, the finding of outstanding infarcts that it has been claimed can be estimated as having taken place during the time of the infection.

It would seem that in the light of present-day serologic and obstetrical data, the A, B, and Rh antigens must be redefined as toxins. When any one or more of these toxins reside in the fetus and can gain access to a mother not possessing them, the symptoms, signs and pathology of pregnancy toxemia supervene. The outcome de-

pends upon the toxicity of the antigen and the reactive properties of the mother. The anatomic structure of the attachment of the fetus to the mother is such that at times a slight blood spill from fetus to mother can take place. When and if this occurs, the property of the antigen as an agglutinogen tends to bring about placental infarction and a sealing of the leak. Any clumping of the comparatively few fetal red blood cells in the maternal blood stream is merely a harbinger of approaching death and dissolution of the fetal cells under antitoxin attack, as obtains with bacteria. The disintegration of the fetal red cells merely increases the toxic content already in the serum. At no time is the picture similar to gross transfusion or to the experiments that conditioned the original definition of the A and B antigens. In the gross transfusion of incompatible blood, the A, B, or Rh antigens involved likely act as toxins as in pregnancy toxemia, but massive agglutination and other hemolytic processes kill the patient before this toxic action can manifest itself. The autopsy manifestations of the toxic action are obscured by the gross agglutination and breaking down of the transfused cells. In pregnancy toxemia, on the other hand, the autopsy findings give a clear picture of the effects of the toxin working over comparatively longer periods of time.

Our time limit on papers does not permit a consideration of the practical application of this material in the management of pregnancy toxemia or erythroblastosis, but it does permit a most important reference to the demands of safe blood transfusion, and the demands of protecting the childbearing possibilities of some women.

If a women is Rh negative, we must always consider the possibility of isoimmunization in even the first transfusion, if she is pregnant or has ever been pregnant. More important still is the responsibility resting upon us all to use Rh similar blood in the transfusion of all females to prevent the possible consequences of isoimmunization. It has been studiously estimated that transfusion is ten times more likely to isoimmunize a woman than is a pregnancy. Failure to follow this injunction may, at any time before menopause, destroy a woman's future ability to bear viable children. The frequency of transfusion is increasing by leaps and bounds in the interests of therapy, and in the fulfillment of these interests its use, as yet, comes far from having reached the saturation point.

The titer data upon which this paper is largely founded resulted from a determined effort on the part of many obstetricians, pediatricians, and general practitioners in the Middle Northwest to solve the problems involved in erythroblastosis fetalis, and safe blood transfusion. As a result, with the serologic aid of Dr. R. W. Koucky, a large pool of antibody titer findings has been formed. This pool of findings has been kept continously open by Dr. Koucky to the inspection of all. Throughout, he has been deserving of the thanks of all involved in giving unstintingly of his serologic experience and advice. The interpretations of the data are those of an obstetrician and, if incorrect, must not be attributed to him.

Conclusions

Genetics decrees that the relationship between fetus and mother is a toxin-antitoxin relationship if the fetus contains toxic antigens not possessed by the mother.

Three of the now known and lettered antigens, namely the A, B, and Rh antigens are, by adequate definition, toxins, when functioning under the above conditions.

In nature, their functioning as agglutinogens marks the beginning of demise and disintegration of the fetal red cells under antitoxin attacks as obtains with bacteria. The liberation of the toxins from the red cells merely augments the strength of the toxins already in the serum.

The symptoms, signs and pathological findings in pregnancy toxemia are caused by one or more of the three blood substances: A, B, and Rh. There may be others, yet unknown.

In erythroblastosis fetalis we are witnessing the destructive action of the antitoxin on the cells from which the toxin arose.

One of the functions of the placenta is to act as a first line of defense in the toxin-antitoxin battle. Due to its anatomic structure, this line of defense is at times broken down, with consequent spilling of fetal serum and red blood cells into the maternal circulation. It would seem that placental infarcts mark the points of leakage, due to the functional capacity of the agglutinin in the antitoxin working upon the agglutinogen in the fetal red blood cells.

(Continued on Page 170)

THE BEDSIDE DIAGNOSIS OF LIVER DISEASE

EDMUND B. FLINK, M.D.

Assistant Professor of Medicine
University of Minnesota Medical School
Minneapolis, Minnesota

IN dealing with patients with obvious liver disease (jaundice or hepatomegaly or similar evidences) the diagnosis can frequently be made at the bedside by the simple expedient of a careful history and physical examination. Furthermore, it is fair to say that a careful history of the illness and a physical examination of the patient must precede all other procedures in the diagnosis of liver disease.

The history should include an orderly inventory of the following facts with complete details of each positive finding. The character, location, radiation and duration of pain and time relationship to meals, relationship to fatty and fried foods, association with jaundice, and association with nausea and vomiting adequately describe one of the most important symptoms. The absence of pain is important, too. The onset, duration, and fluctuation of jaundice and its association with pruritus, relation to the color of the urine and feces, to pain, to the use of toxic drugs such as sulfanilamide, cincophen, dinitrophenol and arsenicals, or to exposure to carbon tetrachloride and trinitrotoluene, characterize the other one of the most important items of the history. The occurrence of the prodromal symptoms anorexia, fever, chills, nausea and vomiting and occurrence of an epidemic of jaundice in the neighborhood are of particular significance in the diagnosis of epidemic hepatitis. Previous plasma or blood transfusions or the use of homologous serum may antedate the onset of hepatitis by thirty to ninety days. A family history of intermittent or persistent jaundice and anemia in many members signifies familial hemolytic anemia in all probability. The amount and duration of the use of alcoholic beverages is important in ascertaining the etiology of hepatic cirrhosis. Intimately related to the alcohol consumption is the question of adequacy of the diet in the past during alcoholic debauches. The occurrence in the recent or remote past of such illnesses as malaria, typhoid, brucellosis, syphilis (and its treatment with arsenicals), amebiasis, and chronic ulcerative colitis may furnish a clue to the etiology of liver disease or of gallstones. As a cause of upper gastro-intestinal hemorrhage bleeding from esophageal varices is common, second in importance only to peptic ulcer. Edema, ascites, weight loss, anorexia and asthenia are common symptoms in advanced cirrhosis.

There are certain features of the physical examination which are of crucial importance in the diagnosis. The appearance of the jaundice and its intensity, the presence of absence of a green tint (or biliverdin icterus), and evidence of pruritus are easily detected by careful inspection of the whole body in good natural light. Considerable jaundice may be overlooked by examination in poor artificial light. The presence of spider nevi over the upper half of the body may be an important clue in the diagnosis of cirrhosis. Spider nevi are small arteriolar hemangiomata with a central pulsating punctum and radial striations extending a few millimeters to as much as a centimeter from the center giving the appearance of a spider. Sometimes the patient is aware of their presence and can tell when each one has appeared, but more often he is ignorant of their presence. They are easily overlooked unless a special search is made for them. Their presence does not establish the diagnosis, but characteristic nevi are presumptive evidence of hepatic cirrhosis. They may occur in normal individuals, during normal pregnancy or diseases of non-hepatic origin. They must be differentiated from the bright red telangiectases without radiations occurring normally over the trunk of adults. Palmar erythema commonly occurs in patients with cirrhosis and nutritional disorders.

The breath of a patient with jaundice should be carefully noted each day. When liver necrosis occurs, there is a characteristic odor termed fetor hepatis or amine odor. It is an aromatic, pungent, sweet odor, which may become so prominent that it pervades the whole room; on the other hand it may be detected only by smelling of the breath directly.

If a patient with jaundice from hepatitis is fasted for a relatively short period of time, hypo-

From the Department of Medicine, University Hospital, Minneapolis, Minnesota.

glycemic episodes may occur with any or all of the symptomatology of an insulin reaction. Stupor, delirium or actual coma may develop independently of hypoglycemia and are of very grave prognostic significance, for there are few patients who recover after becoming comatose. The abdominal examination offers certain helpful and occasionally absolutely diagnostic signs. The presence of ascites is not difficult to elicit when a moderately large amount of fluid is present in the abdominal cavity. The most sensitive index of free fluid is the presence of shifting dullness, i.e. with dullness to percussion over the dependent parts of the abdomen regardless of position. When enough fluid is present, it is possible to identify solid masses by the maneuver of "dipping"; a sudden but, nevertheless, gentle dipping with the fingers of one hand at the level of dullness will give a sensation of splashing fluid against a solid mass whenever one is present. The phenomenon is evidence for free fluid as well as for a tumor mass. Of course, there are many causes for ascites, but one of the most important causes is liver disease and especially cirrhosis. Ovarian cystomas of large size frequently cause confusion with large amounts of free fluid in the peritoneal cavity. The absence of shifting dullness and the presence of flatness in the midline of the abdomen with the patient in the supine position are the most important differentiating features of ovarian cystomas. Pelvic examination may fail to reveal any definite connection of a mass to the uterine adnexae. The rectum must be examined in every patient and it is often possible to detect a "rectal shelf" in patients with carcinomatosis of the peritoneum causing ascites.

Palpation of the liver is easy when it is moderately or markedly enlarged. Careful notation of its consistence, surface, edge, and extent below the costal margin in the mid-clavicular line are important. The edge of a normal liver can often be palpated on deep inspiration. The daily observation of the size of the liver including its upper margin of dullness anteriorly is important in hepatitis. If the liver suddenly decreases in size and continues to do so, and if there is a fetor hepatis and disturbed sensorium, the prognosis is very poor.

The presence of signs of localized peritonitis in the right upper quadrant in association with fever, chills, nausea and vomiting, fatty food intolerance and colicky pains is practically diagnostic of acute inflammatory gall-bladder disease or an acute recurrence of chronic gall-bladder disease. In such patients jaundice usually is evidence of common duct obstruction from stone. An inflammatory mass may be palpated below the margin of the liver in many instances especially when the acute disease is subsiding. Generalized peritonitis may develop as the result of rupture of an acutely inflamed gall bladder with stones.

One of the most neglected and the least appreciated findings in liver disease is splenomegaly. In cirrhosis, acute hepatitis and acute liver necrosis, the spleen frequently is enlarged enough to be palpable. The presence of splenomegaly in a jaundiced patient definitely points toward primary liver disease as a cause of jaundice. Of course it may be enlarged in biliary cirrhosis, may be present as an independent finding in an unrelated but concomittant disease, or the patient may have a familial hemolytic jaundice or anemia. Suffice it to say that the presence of a palpable spleen is evidence of an abnormal state, and one must think strongly of liver disease.

One sign, when present, has more diagnostic importance than any other. In a jaundiced individual the presence of a palpable, non-tender, smooth gall bladder is practically pathognomonic of carcinomatous obstruction of the common bile duct anywhere from the entrance of the cystic duct to the ampulla of Vater. When a gallstone obstructs the common bile duct, the gall bladder seldom is palpable because of previous inflammation with thickening of the gall-bladder wall which prevents distention. A previously normal gall bladder can distend readily. Courvoisier recognized these facts many years ago. Failure to palpate the gall bladder does not exclude carcinomatous obstruction. A distended gall bladder in a non-jaundiced individual is known as hydrops of the gall bladder and must not be confused with the occurrence in a jaundiced individual. This is due to cystic duct obstruction by a gallstone usually. The gall bladder may be palpated when it has a very large stone or many large stones in the fundus. Carcinoma of the fundus of the gall bladder causes an irregular hard mass. Inflammatory masses are usually tender.

A differential diagnosis of liver diseases and diseases of the extra-hepatic biliary tract is possible from the observations made at the bedside and often a definite diagnosis can be made.

Jaundice of the retention type occurs in two diseases. In constitutional hepatic dysfunction there is a very mild jaundice. Usually other members of the family also have mild jaundice. No systemic symptoms and no anemia occur. The spleen is not palpable. In familial hemolytic anemia or icterus the patients are mildly or severely anemic from birth. Jaundice fluctuates greatly. The spleen is always enlarged and usually palpable. One can often get the history of exacerbations of symptoms with chills and fever often called hemolytic crises. Gallstones form in a large number of these patients so it is entirely possible to have the history of typical gallstone colic superimposed upon or even overshadowing the primary complaints thus making the differential diagnosis more complicated. The acquired type of hemolytic anemia with jaundice may be secondary to other diseases such as blood dyscrasias or liver disease, but may also be primary. The spleen is palpable and the anemia is evident, but there is no family history of similar illness except by rare coincidence, and the illness does not date back to infancy or early childhood.

The largest group of jaundiced patients have a regurgitation type of jaundice and it is of utmost importance to establish whether or not there is extra hepatic biliary obstruction or parenchymal liver disease. The urine is beer-brown in color and the stools light to acholic. The liver is enlarged but there is no splenomegaly in simple extrahepatic obstruction of the bile ducts unless the jaundice has persisted for a long time (many months to years). It is necessary to differentiate between cancerous obstruction and calculous obstruction in the group of cases with extrahepatic obstruction.

The commonest type of jaundice (except during an epidemic of hepatitis) is that due to gallstone obstruction of the common bile duct. Characteristically the jaundice fluctuates in intensity, is abrupt in onset and is less intense than in carcinomatous obstruction. The stools may or may not be acholic. Colicky pain which is severe and radiates from the right upper quadrant of the abdomen to the region of the right scapula and interscapular region is present at some time during the early course of illness and usually before the onset of jaundice. Nausea and vomiting frequently accompany bouts of pain. The jaundice deepens perceptibly when there has been an attack of pain unless the stone was passed into the duodenum or shifted in position. The past history of definite intolerance for fatty and fried food, cabbages, onions and especially the history that food actually induces typical gallstone colics are very helpful. Obstruction due to gallstones may be "silent" in so far as an association with pain is concerned. Observation of fluctuation in the intensity of jaundice, in the color of the stools, episodes of chills and fever all suggest the presence of gallstones. It may be impossible to differentiate the "silent" type of gallstone obstruction from acute hepatitis.

Cancerous obstruction is characteristically painless, but there are a number of patients who have vague deep-seated epigastric pain or even pain suggesting gallstone colic. The jaundice is intense, persistent, associated with pruritus and usually abrupt in onset. Usually there are no chills, fever or vomiting. The gall bladder is frequently palpable below the liver margin and is not tender. This finding is of the greatest importance, for it is pathognomonic of cancerous obstruction of the extrahepatic biliary tract in the jaundiced patient. Carcinoma of the gall bladder and bile ducts usually occurs in patients with gallstones too so any or all of the symptoms of chronic cholelithiasis may be present in the past history or the present illness. Carcinoma of the gall bladder may cause a great deal of pain but causes jaundice only in the cases where it invades the common bile duct.

Rarer forms of obstructive jaundice are due to lymph-node enlargement at the porta hepatis causing compression of the hepatic or common bile ducts. Hodgkins disease, lymphatic leukemia, lymphosarcoma or carcinomatous metastases may be the primary disturbance. Recognition of lymphoblastomata is aided by finding generalized lymph-node enlargement and moderate to very marked splenic enlargement.

Stricture of the common bile duct is a serious complication of cholecystectomy in which the duct was inadvertently ligated, cut, or injured. The history usually reveals that jaundice began shortly after operation and persisted, that a large amount of bile suddenly appeared at the wound as if from an abscess which ruptured, bile continued to drain indefinitely from the wound, and jaundice appeared or deepened as soon as the bile fistula closed. Strictures frequently are not treated im-

mediately, so biliary cirrhosis with hepatomegaly and splenomegaly develop.

Regurgitation jaundice of hepatic origin is frequently encountered. The onset of jaundice immediately after exposure to toxic chemicals or drugs such as carbon tetrachloride, cincophen, dinitrophenol, arsenicals, sulfanilamide, benzene, etc. suggest the specific chemical as the etiological agent. The history of exposure to patients with epidemic hepatitis, transfusion with human blood or blood derivatives, the use of homologous sera of any kind in the recent past directs one's attention to acute hepatitis which probably is due to a filterable virus or viruses. The history of prodromata including fever, severe anorexia, nausea and vomiting, malaise, and bowel disturbances before the onset of jaundice is very suggestive evidence in favor of acute hepatitis. There are authentic cases of acute hepatitis without jaundice and these are very difficult or impossible to recognize unless an epidemic is occurring. The spleen is palpable in about one-half of the cases and undoubtedly the percentage is higher when daily examinations are made. The size of the liver should be noted on initial examination and checked frequently during the course of illness. Observation of the breath for a fetor hepatis and observation of the sensorium have been emphasized above. Daily observations of the feces for the appearance of bile-pigment is important for the recognition of the recession of jaundice. Ascites may be present in the more severely ill patients.

Recognition of hepatic cirrhosis is dependent on the finding of some of the characteristic features such as a firm, finely nodular liver, enlarged spleen, spider nevi, ascites, evidences of collateral circulation on the abdominal wall and evidence of malnutrition and jaundice. Probably not more than half of all of the patients are jaundiced and any of the above-mentioned features may be missing. The skin is soft and the axillary hair is often sparse. When the skin is deeply pigmented in a male who is also diabetic, one should suspect hemachromatosis or pigment cirrhosis.

The liver may be enlarged as a secondary manifestation of other diseases. Congestive heart failure causes chronic passive congestion of the liver. The liver frequently is tender to palpation, and not infrequently the patient may have pains in the right upper quadrant of the abdomen as a result of the congestion. Metastases to the liver may cause massive enlargement of the liver. One can recognize metastatic nodules in the liver with reasonable certainty if one can palpate a depressed area in the center of the nodule. The liver often is tender when it is filled with metastases. Recognition of symptomatology and physical characteristics of the primary carcinoma is very important. There are many other diseases which may affect the liver secondarily, but these will not be discussed separately.

Summary

The importance of the clinical examination of the patient with evident or with suspected liver disease cannot be overemphasized. There is often a tendency to turn quickly to laboratory aids without adequate clinical examination. However, the so-called liver function tests may help and may confirm a clinical diagnosis or indicate the need for more prolonged observation and study of a case. Admittedly there are times when the clinical picture is so complex that an exact anatomic diagnosis of the liver disease can be made only by some type of surgical procedure. Careful interpretation of the details of the history and of the physical examination will often, probably in the majority of cases, direct the doctor to the correct diagnosis.

WHY DO AIR TRAVELERS CHEW GUM?

People making their first trip by air are surprised to have the hostess offer them gum to chew as the plane lands. The reason for the gift of gum, says the Sonotone research laboratories, is that the working of the jaws in chewing tends to keep the air pressure equalized on both sides of the ear drum. Chewing action opens the throat end of the Eustachian tube, which is an air duct leading to the middle ear. In landing, an airplane moves from low to high atmospheric pressure, a situation which would cause discomfort if the increased pressure was exerted only on the outer side of the ear drum.

KERION: A REVIEW

CARL W. LAYMON, M.D.
Minneapolis, Minnesota

IN modern medical writing the term kerion is used to designate an acute, highly inflammatory, carbunculoid type of fungous infection which predominantly involves the scalp and bearded region. The word is an extremely old one in medical nomenclature, the ancient Greeks having used it to denote deep, infiltrative, cutaneous inflammations. Kerion is not unusual in this part of the Midwest because a fair proportion of the population is engaged in farming or dairying, occupations which necessitate close contact with animals which are subject to fungous infections due to species which cause this type of reaction.

Kerion can be produced by several species of fungi. Lewis and Hopper[3] reported this form of infection due to Trichophyton gypseum, Microsporum lanosum, M. audouini, M. fulvum, T. crateriforme and T. niveum. The great majority of cases are produced by species of fungi which are highly pathogenic to animals (zoophilic fungi of Wise and Sulzberger[9]), especially T. gypseum. It is extremely rare that the so-called human pathogens (anthropophilic fungi of Wise and Sulzberger) evoke such a reaction.

Clinical Features

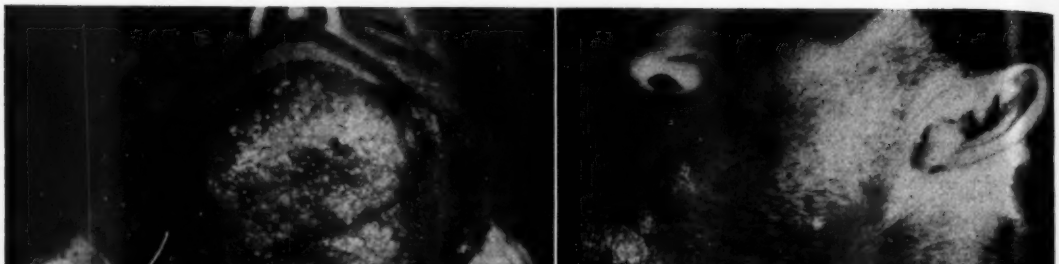
At the beginning kerion rarely presents the granulomatous tumefactions which are so characteristic of the disease at the peak of its intensity. It usually starts as an ordinary patch of squamous or vesicosquamous ringworm on a hairy area or which has spread to a hairy area from a neighboring portion of the glabrous skin. In some instances the early lesion resembles an ordinary, small furuncle. Frequently there is but one lesion and rarely more than a few. Within a short time the plaques become larger and more inflamed and the surface becomes covered with impetiginous crusts. After a few days, or at the most two or three weeks, follicular pustules, either few or in large numbers, predominate the picture. This pustular, follicular infection spreads to the perifollicular tissues and through confluence of the adjoining follicular infiltrations, the entire lesion becomes a boggy edematous mass. In this fully developed stage the lesion is a rather sharply demarcated, sloping bordered, macaroon-like, spherical tumor of red or livid color which, for a time, is firm and later mushy due to coalescence of the follicular abscesses. At this point the surface is oozing and the follicular pustules, when broken, emit sanguinous pus. The odor is repulsive. The individual round lesions increase in size but rarely grow larger than 6 or 7 cm. in diameter. They are usually raised from 0.5 to 1 cm. above the plane of the surrounding skin. The first lesion is usually the largest and these which appear later rarely become as large, although sometimes there may be 6 or 8 kerions on the scalp or bearded region which are of almost equal dimensions. The skin between the lesions is usually normal or presents only a scattering of follicular pustules which occasionally coalesce to form small nodular foci. There is considerable variation in the intensity of the inflammation in different cases.

After the inflammation has reached its height the pustules evacuate their contents and the affected hairs are expelled. At this stage the surface becomes smooth and hairless and the epidermis thin and tense. Extensive erosions and even ulcerations frequently develop and the gaping follicular openings give the lesions a "worm-hole" appearance.

Early in the course of kerion the hairs are unbroken and appear normal. Stubby hairs, which are so frequently noted in infections due to anthropophilic fungi, are rarely seen in kerion. Later, when the phase of purulent folliculitis is fully developed, the hairs can be extracted easily without breaking them. Apparently healthy hairs, situated between the grossly diseased ones, can sometimes be pulled out without difficulty, the root being atrophic as a result of the neighboring inflammation. Even in the final stages, after expulsion of most of the hairs, a few tufts of healthy hair may remain.

The average duration of kerion is six or eight weeks, although severe cases may last for three months. There is a constant tendency toward spontaneous healing. The hair almost always regrows and there is an eventual return to complete

From the Division of Dermatology, University of Minnesota, H. E. Michelson, M.D., Director.



KERION OF THE BEARD IN A FARMER.

normalcy, although permanent baldness may occur rarely.

Besides the aforementioned local changes, systematic symptoms are commonly noted as a result of absorption of substances into the lymphatics and blood stream. In kerion of the scalp a retro-auricular, occipital and cervical adenitis is an almost constant finding and lymph nodes even farther removed from the primary focus may be enlarged. The nodes are at first firm and tender; later there may be softening and rupture to the surface. Fatigue, malaise, and cephalgia have been observed frequently. The fever may be high in cases of extensive kerion. Miescher[5] reported cachexia in extremely severe cases. The blood usually shows a polymorphonuclear leukocytosis.

Kerion of the beard, or sycosis parasitaria, is analagous to the disease as it occurs on the scalp. Beginning as superficial, vesicular, discoid or ringed foci, a pustular folliculitis soon develops and the entire lesion becomes edematous and raised above the surrounding skin. As a rule there are only a few foci; sometimes only one surrounded by small abortive lesions. Rarely, new superficial vesicular foci develop side by side with the granulomatous masses and nodules. As on the scalp the skin between the lesions is usually normal. The chin and sides of the face are the most common sites of involvement; rarely the upper lip is affected. The general symptoms and course parallel those of kerion of the scalp.

Kerionic lesions occasionally occur on areas other than the scalp and bearded region presumably due to infection of lanugo hairs by ectothrix trichophytons. Such lesions have been noted on the dorsums of the hands, forearms, sides of the neck, buttocks, inguinal region and external ear. The term "agminated folliculitis" has been used to designate this affection.

Granuloma trichophyticum should also be mentioned in relationship to kerion. It is a form of deep, infiltrative trichophytosis first described by Majocchi[4] and discussed chiefly in the Italian literature. It is characterized by round, smooth, indolent, bean- to nut-sized nodules which are tender on pressure, and usually light red in color. There is a tendency toward grouping. Early in the course of the disease the nodules have an elastic consistency but later become soft and almost fluctuant. In some cases, however, they become hard and keloidlike. There are no signs of suppurative folliculitis or abscess formation as in kerion, a point which was stressed by Majocchi and subsequent writers. The softening which occurs in the lesions represents a secondary necrobiotic change in the granulation tissue similar to that which occurs in gumma. On pressure a serosanguineous material rather than pus can be expressed. Impetiginous changes and pustules at the follicular orifices occasionally occur late in the disease but are regarded as a secondary infection by pyogenic cocci.

The nodules don't develop primarily but become superimposed on a focus of ordinary ringworm almost exclusively produced by anthro-

pophilic fungi. The lesions are usually located on the scalps of children from six to twelve years of age or on the bearded regions in adults. Rarely they develop from squamous lesions of the glabrous skin.

The course of granuloma trichophyticum is extremely torpid, the disease usually lasting several months. In time the granulation tissue resorbs or softens, and sometimes degenerated material is discharged on the surface. The skin may return to normal or become somewhat atrophic.

There has been considerable discussion concerning the mode of entry of fungi which are found in the granulation tissue in the cutis. Although some authors have stated that the fungi penetrate from the surface directly into the cutis, the concensus is that the follicles represent the point of entry since, in the course of the affection, the diseased hairs and follicles become degenerated permitting the resorption of fungus elements into the cutis. Sabouraud thought that there was no good proof that there was any active emigration of fungi from the surface to the cutis. Disintegration of the follicle, whether as a result of purulent inflammation or degeneration, thus plays the principal role in the development of granuloma trichophyticum. Majocchi and Mazza tried to introduce fungi directly into the cutis in an effort to produce this type of the disease experimentally, but the results were practically negative. Following intracutaneous implantation of fungi in humans and dogs, nodules or abscesses rapidly formed followed by expulsion of the fungi. Similar attempts in cock's combs produced infiltrated plaques and nodules after 3 to 4 weeks which involuted slowly and showed tuberculoid structure histopathologically. Majocchi, however, did not believe that the lesions were analogous to those in the human as the course was much more rapid and the tendency to heal much greater.

So-called chronic kerion represents a slowly progressive gummoid change of the contents of ordinary kerion and is always preceded by an acute stage with pustular folliculitis.

In those cases of granuloma trichophyticum in which trichophytin tests were done, the reactions were weakly positive in contrast to the strongly positive reactions which characterize kerions.

As far as other diseases are concerned the differential diagnosis of kerion is usually not difficult. The prominent feature of pustular folliculitis is against other granulomas such as syphilis, tuberculosis, actinomycosis, blastomycosis and mycosis fungoides. The relatively sharp demarcation of the lesions aids in the differentiation from pyoderma, though in its early stages kerion may resemble impetigo. In the former, however, tiny blisters and pustules may be seen as the border while the individual lesion in impetigo is a bulla which spreads peripherally.

On the face there may be confusion with staphylococcic sycosis in the early phases of kerion. One of the chief characteristics of the latter, however, is the agmination of small lesions to four larger nodules, a feature not seen in ordinary sycosis. The presence of round and annular vesicular foci characterizes the initial stages of kerion but not coccygenic sycosis. The latter affects the upper lip with great frequency while kerion almost never occurs in that location.

Chancre of the face is not apt to be confused with kerion since there are no follicular pustules in the former.

Demonstration of the causative fungi in wet preparations and cultures affords a definite proof in the final diagnosis of kerion.

Examination of Hairs and Pus—Cultures—Inoculations

Sabouraud stated that it is a curious but certain fact that the examination of pus from kerion almost never shows recognizable fungi, yet cultures from the pus are usually positive and are regarded as the easiest and surest proof of the nature of the disease. This is explained by the fact that the dissociated fungous elements are so easily confused with cellular debris. In fully developed kerion it is almost impossible to demonstrate fungi along the hairs in potassium hydroxide preparations although such examinations are positive at the very beginning of the lesion. As previously mentioned the vast majority of cases of kerion are due to Trichophyton gypseum. The growth of the organism is largely on the outside of the infected hair which becomes covered by a mantle of mycelial elements and spores. The latter are 2 to 4 mm. in size and, in wet preparations of potassium hydroxide, appear to have the same mosaic arrangement which characterizes

the microsporums. If the slide is pressed a little bit, however, it can be seen that the spores of T. gypseum are arranged in chains, a feature never seen in microsporum infections.

Inoculations into the skin of both humans and animals is not difficult. Sabouraud spoke highly of Rivalier's procedure in which a paste containing a mixture of honey and culture powder is applied to a hairy part of the skin. The inoculation is usually positive by the seventh day. The hair of the monkey illustrates the microscopic features of T. gypseum in hair more beautifully and schematically than human hair. Sabouraud stated that primary cultures should be used for inoculation, since in later cultures the parasitism is changed and sporulation is lost.

In animals the infection is less intense and suppurative than in man. Discharge from the lesions resembles large drops of glue which exudes from between the hairs and mats them down.

Histopathologic Features

The histopathologic findings in kerion vary considerably in extent and intensity. In the beginning inflammatory changes in the epidermis are noteworthy, and the tendency to follicular localization is evident early in the course of the disease. Superficial epidermal spaces filled with leukocytes may be seen and there is a perifollicular infiltrate made up of lymphocytes, plasma cells and polymorphonuclear leukocytes. By means of the gram stain mycelial filaments may be demonstrated in the horny layer of the follicles and around the hairs. As the disease progresses the inflammation spreads toward the hair root although the latter is the last part of the hair to become affected and may be entirely spared. The luxuriant growth of fungi about the hair shafts has, in Miescher's opinion, much to do with the high degree of allergy in kerion. The tissue reaction is so exudative that small abscesses may develop in the middle or upper portions of the follicle. The infected hair finally loosens and falls out, leaving only the root. The sebaceous glands associated with the follicle are also involved early and after a stage of leukocytic invasion the entire structure disintegrates.

The follicle walls become edematous, vacuolated and permeated by leukocytes. The degenerated cells stain poorly. Lymphocytes, plasma cells, and eosinophils also make up part of the infiltrate along with large macrophages, large nucleated fibroblasts and epithelioid cells. The perifollicular vessels are dilated and filled with leukocytes. After complete degeneration of the follicle wall spherical abscesses may be seen in the cutis, some of which contain bits of disintegrated hair and fungi. Degenerated filaments and spores may also be seen lying free between the leukocytes. No other micro-organisms are in evidence and cultures of pus show single colonies of staphylococci only exceptionally. The inflammatory and degenerative changes in kerion seem out of all proportion to the number of fungi seen in the tissues.

In severe cases the abscesses penetrate to the surface, usually through the follicular orifices and only rarely through degenerated intrafollicular portions of the skin. In less severe cases the abscesses are gradually absorbed. Late in the course of kerion giant cells, both the foreign body and Langhans types, may be seen along with epithelioid cells and fibroblasts. Portions of the degenerated follicle remain as irregular epithelial masses. Another feature which has been observed is the formation of pseudoepitheliomatous changes of such degree that differentiation from true epithelioma may be difficult. In these cases irregular karyokinesis and pearl formation may even be present, adding to the confusion. Fully developed kerion presents a variegated and complicated picture. Not all adjoining follicles are affected and those which are involved become so at different stages of the disease. Some follicles remain completely intact throughout the entire course.

Because of the degenerative changes in the follicles and the formation of granulomatous infiltrations as deep as the subcutaneous blood vessels, the entire architecture of the cutis is changed. The elastic and collagenous fibres diminish to mere remnants. The sweat glands may be seen unchanged in the center of a granulomatous mass. Usually epithelial sprouts may be seen in the upper part of the follicles which later take part in the process of regeneration. These sprouts, along with follicles which escape the inflammation, explain why permanent baldness is rare.

In granuloma trichophyticum the tuberculoid structure of the infiltrate is outstanding as compared to the acute pustular folliculitis which predominates the picture of kerion. In the former, the middle and deep portions of the cutis are involved by such nodular infiltrations. The infil-

trate is perifollicular and the follicles are often cystic and degenerated to the point of being unrecognizable. Fungus containing fragments of hairs lie free in the granulation tissue. Fungi may also be seen between the cells or enclosed in giant cells. In the unusual fibroid form of granuloma trichopyticum fibrous masses are present in the cutis. Suppurative changes in this form of fungus infection are much less noticeable than in kerion.

Allergy in Kerion

Bloch[1] studied the biologic and allergic phenomena in fungous infections extensively and he and Jadassohn and their followers contributed much of the original work in this field, much as Koch and his school did in tuberculosis. Although many species of animals have been used in experimental work on pathogenic fungi, the guinea pig has remained the principal animal and Achorion quinkeanum the principle fungus in studies concerning diseases produced by Hyphomycetes. Sulzberger[7] stated one strain of A. quinkeanum has been used in Zurich for over 30 years and has regularly produced uniform experimental results.

Primary infections in the guinea pig run their course in three or four weeks with the peak of intensity about the twelfth day. In reinoculations the response is accelerated, being most intense within six days and completely healed in ten or twelve days.

Similar experiments in man strongly resemble those in the guinea pig as far as the difference between first inoculations and reinoculations are concerned, except that in man the course of events is less constant and regular. Differences in individuals and variations in the response at various inoculation sites are greater in man. Variations in the response to various strains of fungi are also more noticeable. In general, the duration of the infection is much longer (two or three months in first inoculations of A. quinkeanum), and the periods of peak intensity and resolution are not so sharply bordered. The length of the inoculation period is also more fluctuant. In man the entire course of events more closely approaches that which takes place in the guinea pig when experimental inoculations are made with fungi of low virulence. In man, the more virulent the strain used for inoculation, the greater is the analogy to the infection in the pig.

In reinoculations in many with zoophilic fungi, whether the first infection was spontaneous or artificial, the reaction is accelerated and the course of the infection is abortive or shortened, influenced by the allergy of the entire skin produced by the first inoculation. This allergy is not type-specific and persists after the primary infection has healed. The degree of allergy depends on the type of fungus, the mode of inoculation, the individual and other unknown factors. Accordingly, the reaction to reinoculations varies from those which closely simulate the first inoculating to accelerated abortive infections in which fungi can be demonstrated to, finally, almost complete allergic reactions in which no fungi can be found. Other factors which influence the degree of allergy and the fate of reinoculations are the depth, extent and number of primary foci, the number of preceding inoculations, and the distance of the reinoculation site from the primary focus. There are undoubtedly other factors too, such as the individual capacity to react to a pathogenic organism and the ability of certain fungi to elaborate allergizing substances. Experimental reinoculations in patients with kerion have been performed by various workers and the results have followed the general rules previously outlined.

In infections in man produced by anthropophilic organisms the degree of skin sensitivity is not high. In kerion, however, and in other infections produced by zoophilic fungi the degree of sensitivity to trichophytin is high. There are exceptions to these general rules, however, which defy explanation.

In kerion, "id" eruptions are not uncommon because of the high degree of sensitivity. In this type of infection especially, the sequence of exposure, primary infection, development of allergy, development of primary lesions and the eruption of the "id" is often clearly apparent. In trichophytids almost every form of allergic reaction of the skin may be represented, although deep granulomatous and necrotic processes seen commonly in tuberculids and syphilids are rare among "ids" due to fungi.

Treatment of Kerion

In discussing the therapy of kerion two points should be brought out: (1) There is an outspoken tendency toward spontaneous healing and (2) the treatment is not as routinized as in infec-

tions due to anthropophilic organisms such as tinea capitis due to M. audouini. Many therapeutic methods have been described in the literature and it is not easy to evaluate them because the favorable end result is inevitable on biologic grounds alone.

The various therapeutic measures may be discussed under the following headings:

A. Local Agents Not Specifically Antiparasitic.—These include soothing, cleansing and softening agents such as moist compresses, moist or dry heat, ultraviolet radiation, incision of small abscesses and currettage of masses of granulation tissue. Dr. Charles Freeman[2] believes that the judicious use of solid carbon dioxide shortens the course of kerion. In most cases mild, moist compresses which soften and remove crusts and facilitate drainage, constitute the best treatment of the disease.

B. Epilation (Manual or By Means of X-rays).—Since the severity of the inflammation in kerion results in expulsion of the diseased hairs epilation by means of x-rays is rarely, if ever, indicated. Manual epilation of infected hairs, along with the removal of crusts, is helpful.

C. Strong Antiparasitic Remedies.—The use of medications containing high percentages of resorcin, sulfur, chrysarobin, mercurials and salicylic acid is rarely necessary. The use of 20 per cent salicylic acid ointment has favorably impressed Ormsby.[6] The internal use of iodine is unnecessary in kerion.

D. Non-specific Stimulation of Immunobiologic Defense Mechanisms.—Intramuscular injections of turpentine in olive oil, injections of milk and the use of intravenous typhoid vaccine have been used by many in the treatment of kerion. The concensus is that such methods are of slight value.

E. Trichophytin Desensitization.—Oppenheim is firmly convinced that trichophytin desensitization is valuable in kerion and other forms of fungous infection. Miescher believed such treatment was helpful in kerion, but of no value in non-inflammatory types of mycosis. In the experience of most observers such treatment has been unnecessary and since untoward local and systemic reactions can result it seems contraindicated in a disease which heals spontaneously.

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POSTMORTEM EXAMINATIONS AND THE JEWISH LAW

Because of the undesirability of some resistances against postmortem examination that appear among the relatives of Jewish patients who die, Dr. I. M. Rabinowitch, special lecturer in medical jurisprudence at McGill University, Montreal, recently investigated the laws with regard to postmortem examination contained in the Bible and in the Talmud. He points out that the attitudes of non-Jewish people of the same period toward postmortem examinations were much the same as those of the Jewish people. Arguments in favor of prohibiting postmortem examinations are based on four authentic instances in which a requested postmortem examination was prohibited by a celebrated rabbi. However, Rabinowitch points out that permission was refused in each instance because of the argument that the postmortem examination would not be of any value either in clearing the cause of death or of any benefit to the living. The thorough search which Rabinowitch has made convinces him that nowhere in the Bible, in the Talmudic, in the post-Talmudic or in the later Rabbinical writings is there any evidence that postmortem examinations are prohibited by Jewish law. Indeed, such prohibition would be inconsistent with the fundamental directing principle in Judaism—"The Laws were given that man shall live by them, not that they shall die by them."—*J.A.M.A.*, 130:151, (Jan. 19) 1946.

URETERAL OBSTRUCTION CAUSED BY ANEURYSM OF THE ILIAC ARTERY

HUGH F. RIVES, M.D. and EDWARD N. COOK, M.D.
Rochester, Minnesota

LAZARUS and Marks[1] have described the urinary symptoms associated with aneurysm of the abdominal aorta. They pointed out that the most frequent causes are arteriosclerosis and syphilis. The most common urographic finding in their cases was renal or ureteral displacement, and they stressed the diagnostic value of lateral pyeloroentgenogram.

We wish to report a unique and unusual case. It is the only such case seen at the Mayo Clinic. A review of the literature, although not completely comprehensive, has not revealed a similar case.

Report of Case

A man, fifty-eight years of age, was first seen at the clinic February 21, 1944. He had been perfectly well until two and a half months previously, when he had had generalized aching, malaise, loss of appetite and a temperature of 102° F. The fever had lasted several weeks and the patient had been treated symptomatically. One month after his illness, severe pain had occurred in the right groin and had extended to the right testis and right renal region posteriorly. The pain had been accompanied by hematuria, pyuria and urinary frequency. Treatment with sulfanilamide for one week had produced some improvement but very severe pain subsequently had occurred in his right groin and right hip. The pain in the hip had seemed to extend posteriorly to the midline of the lower part of the back. Urologic investigation was said to have disclosed bilateral pyelonephritis and two small stones in the left kidney. Urinary culture had revealed Salmonella aertrycke. Treatment with sulfanilamide had been continued and the fever and pain had subsided gradually in about ten days. During the next three weeks the patient had had intermittent fever and pain in the right groin. At the end of this period, he came to the clinic. The patient was well developed and well nourished but appeared moderately ill. The lungs and heart were normal. The blood pressure was 104 mm. of mercury systolic and 54 mm. diastolic. The pulse rate was 88 and his temperature was 101° F. The abdomen was slightly distended and there was some tenderness in the suprapubic region. Rectal examination revealed a questionable nodule in the lower pole of the right lobe of the prostate gland. It was felt that the patient possibly had carcinoma of the prostate.

Microscopic examination of the urine revealed hematuria, grade 1 (on the basis of 1 to 4), and pyuria, grade 2 (35 cells in each microscopic field viewed under the high-power objective). Urinary culture disclosed Salmonella. The value for the hemoglobin was 9.1 gm. per 100 c.c. of blood. The erythrocytes numbered 3,580,000, and the leukocytes numbered 6,600 per cubic centimeter of blood respectively. Flocculation tests for syphilis were negative. The value for the blood urea was 26 mg. per 100 c.c.; the value for the alkaline serum phosphatase was 7.0 units (King-Armstrong) per 100 c.c., and the value for the acid serum phosphatase was 1.0 unit per 100 c.c. The sedimentation rate of the erythrocytes was 93 mm. in one hour (Westergren). A roentgenogram of the thorax revealed torsion of the aorta. Anterior, posterior and lateral roentgenograms of the lumbar vertebrae did not reveal any abnormality. An excretory urogram disclosed delayed excretion on the right side and dilatation of the renal pelvis and calices and upper part of the ureter. The cause of the obstruction was not evident. Urography did not disclose any abnormality of the left kidney except a small cluster of shadows (Fig. 1). The cause of these shadows could not be determined. At this stage of the examination, it was felt that the patient probably had a carcinoma of the prostate which had caused obstruction of the right ureter. On February 23, 1944, cystoscopy revealed a trilobar prostatic enlargement, faint trabeculation of the bladder and erosion of the right ureteral orifice. A No. 5 ureteral catheter could be passed easily up the right ureter although it seemed to fit snugly as it was being passed through the portion of the ureter corresponding to the wall of the bladder. A catheter could be passed easily up the left ureter. Rectal examination was repeated and the prostate was thought to be the site of a malignant lesion. Repeated attempts to obtain a right pyelogram were unsuccessful. A left pyelogram did not disclose any abnormality.

Fig. 1. Excretory urogram showing dilatation of the renal pelvis and calices and upper third of the ureter on the right side.

On March 25, 1944, two days after cystoscopy was performed, the temperature was 103.2° F. There were no physical findings to explain the elevation of tem-

Dr. Rives is a Fellow in Urology, Mayo Foundation; Dr. Cook is a staff member in the Section on Urology, Mayo Clinic, Rochester, Minnesota.

Fig. 2. Aneurysm of the right common iliac artery with clot in place.

perature. The patient complained of pain in the right groin and hip, and at this time he began to complain of weakness in the right leg. The fever subsided in the next few days. On March 1, 1944, pitting edema of right lower extremity was noted. Arterial pulsations were normal and the temperature of the skin of the right leg was increased. A consultant made a diagnosis of thrombophlebitis of the right iliofemoral vein and suggested that a pelvic lesion might be pressing on the femoral vessels and causing the thrombophlebitis.

On March 27, we were able to palpate a definite mass in the right lower quadrant of the abdomen. There was rigidity of the right rectus in the lower half and tenderness was present over the entire right lower quadrant and in right flank. During the day, the patient complained of severe pain in right side of abdomen. At 11:30 p.m., he complained of severe pain in right side. Shock developed and the patient died.

Necropsy revealed an arteriosclerotic aneurysm of the right common iliac artery (Fig. 2) which had ruptured and caused extensive retroperitoneal hemorrhage. The aneurysm had caused partial obstruction of the right ureter, hydronephrosis, grade 2, and beginning pyonephrosis. The ureter was compressed between the right common iliac artery and the aneurysm at the point of crossing the artery. The patient also had one stone, 2 mm. in diameter, in lower calix of the left kidney, arteriosclerosis, grade 3, of the aorta and coronary sclerosis, grade 1.

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116 PHYSICIANS DIE IN ACTION DURING 1945, JOURNAL REPORTS

Death Claims Approximately 4,015 Doctors as Compared with 3,415 During '44; Heart Disease Leading Cause

Death claimed approximately 4,015 physicians during 1945 as compared with 3,415 the previous year, according to the January 19 issue of *The Journal of the American Medical Association.*

On the basis of an analysis of 2,962 obituaries published in *The Journal* during the year, it was found that the average age at death was 65.3, the same as in 1944. The majority of the physicians—494—died between the ages 70 and 74.

Heart disease continued to lead the causes of death among physicians. Coronary thrombosis and occlusion accounted for 655 deaths, 246 occurring between the ages 60 and 69. Cancer and tumors accounted for 296, and pneumonia claimed 145 doctors.

Of the 25 suicides recorded, 11 were the result of bullet wounds, three of a cut artery and three of drugs. There were 87 accidental deaths and 33 of them involved automobiles.

Among the decedents were 256 who had been teachers in medical schools, of whom 97 had reached professorial status. There were eight deans, one of whom had also been president of a medical college, two principals of a high school, two teachers in public schools, one of whom had been head of the business and practice department of the city's high schools, and 78 members of education boards. One hundred and forty-four had been health officers.

The 2,962 *Journal* obituaries included 116 physicians who were killed in action during World War II and 118 who died while in military service. Nineteen physicians lost their lives in the Pacific area, 13 in Germany, 12 each in the European Theatre of Operations and in France, and 11 in Okinawa, including six who were lost in the attack on the *Comfort.*

Of the 118 physicians who died while in military service, aircraft accidents caused the deaths of 22 and vehicle accidents of 10. Heart disease accounted for 25 deaths, 12 of which were attributed to coronary thrombosis. Cancer caused 13 deaths.

The 116 deaths classified killed in action give an average age of 33.0, while the 118 who died while in military service average 40.1. By combining the two, an average age of 36.6 is noted.

Of the seven U. S. Public Health Officers whose deaths were recorded, one died when he slipped on the Jacob's ladder while assisting wounded aboard ship and one when his ship was destroyed in enemy action.—*A.M.A. News,* Jan. 17, 1945.

NOTES ON THE HISTORY OF MEDICINE IN HOUSTON COUNTY PRIOR TO 1900

By NORA H. GUTHREY†
Mayo Clinic
Rochester, Minnesota

(Continued from January issue)

J. M. Riley, who was a native of White County, Illinois, was born on January 18, 1833. A graduate of the Eclectic Medical Institute at Cincinnati, Ohio, in 1859, he came in the following year to Brownsville, Houston County, Minnesota, where he announced himself as physician and surgeon and where he remained, through the boom days of the village and into the period of its decline, at least as late as 1899, according to an old gazetteer and business directory. Dr. Riley was a Mason. In 1870 he served as county coroner and in 1879 he was elected from the first district as a representative in the state legislature. After 1883 he practiced medicine under a state exemption certificate.

Captain See was discussed fully in paragraphs about Brownsville Township, in the narrative preceding this series of biographical notes. Supposedly the possessor and user of medical knowledge, and said to have been in Brownsville during or after the Civil War, he proved on investigation to have been not a physician, but Captain Charles H. See, of Caledonia, Provost Marshal and President of the Enrollment Board of the First Congressional District, which included Houston County. He undoubtedly was at times in the county in the course of duty, and inasmuch as his fellow members on the board were physicians and because much of the work of the board had to do with claims for exemption based on physical disability, there is reason for local mention of Captain See as a physician.

Edwin M. Sheldon, of the regular school of medicine, came from Illinois in 1881 and for about two years practiced medicine in the village of Houston. In 1882 his office was in Field and Briggs' building and he was boarding at the Sherman House. It appears likely that he and Dr. Timothy A. Pope were removing from the village at about the time that Dr. H. P. Johnson settled there. Although Dr. E. M. Sheldon may have been related to the branch of the Sheldon family to which Dr. G. J. Sheldon belonged, such assumption has not been borne out by information at hand.

Giles James Sheldon, tall, slender, blue-eyed, mentioned often in these pages, was one of the earliest settlers and, it is believed, the first physician of Houston County, who arrived on June 27, 1853, at Brownsville, in Brownsville Township.

He was born at Norway, Herkimer County, New York, on May 12, 1806, the son of Caleb Sheldon, a farmer born of English parents, and Rebecca Giles Sheldon, of English descent, a native of New England. In his genealogy

of the Sheldon family for four generations Dr. Sheldon mentioned that three Sheldon brothers, who were English seamen, came to America to escape King Charles II; their descendants became farmers. In his immediate family he referred to only one brother and one sister, G. W. Sheldon and Clotilda.

A frail child and youth, Giles J. Sheldon received his early education at home from his mother, who gave him excellent grounding for his subsequent cultural and scientific training. When he was fifteen years old, he entered Fairfield Academy, at Fairfield, New York, on February 5, 1821, and there studied as his health permitted. On February 9, 1826, he entered Lowville Academy, at Lowville, in the state, and the following year, he enrolled at Union College in Schenectady. In June of 1829 he registered at Camden Academy, at Camden, New York, but returned to Union College, where he was elected to the Phi Beta Kappa fraternity and where, in 1830, he received his degree of Bachelor of Science. For two years he studied theology with the expectation of becoming an Episcopal clergyman, but, influenced by his uncertain health, he transferred to the study of medicine. During the years from 1831 to 1833, inclusive, he studied botany, chemistry and medicine at the College of Physicians and Surgeons of the University of the State of New York, at Fairfield; he received instruction in anatomy and physiology from James McNaughton, M.D., and P., and on completion of his work with Dr. McNaughton received certificate No. 165, dated October 30, 1833. At the beginning of the following year, on January 21, he was graduated as Doctor of Medicine. There is record in his private papers that he studied also under several physicians in New York and in Philadelphia, among whom were Dr. Loon (?) and Dr. Booth, and under Dr. Willoughby, of Newport, and that in the intervals of studying medicine he was engaged in farming and surveying.

Dr. Sheldon began medical practice in Norway, New York, in February, 1834, but late in June of that year he removed to Trenton, in Oneida County. On April 10, 1835, he was appointed postmaster at Holland Patent, New York, and served until he moved to Van Buren Harbor, New York, where he practiced medicine in 1836 and 1837. On December 2, 1835, he had been appointed surgeon for the Fourth and Fifth Brigades of Riflemen of the State of New York, an appointment in which he was qualified on March 30, 1836. In 1837 he traveled to Illinois, a state that was the field of his medical practice for three years. In 1840 he moved to Ohio and for the first two years practiced at Amanda. From December 3, 1841, to April 24, 1849, he was in Mercer County, practicing medicine and, for the latter part of the period, living in the village of Mercer and studying law in addition to carrying on his medical work. Thereafter, in Celina, Mercer County, he combined his study of law with his other pursuits to such purpose that he was admitted to the bar on August 5, 1852. On April 20, 1853, he left Celina with his family for southeastern Minnesota in the hope of improving his health, which never had been robust.

Giles James Sheldon's first marriage was to Elizabeth Cullum. His second marriage was to Margaret Roberts, of Celina, Ohio, a native of the state, of Welsh and Swiss descent. Of the second marriage there were seven children: James Giles Sheldon, a railway engineer; Elizabeth Sheldon (Mrs. Reuben) Hoag, a teacher; Celina Sheldon (Mrs. H. J.) Herzog; Henry Robert Sheldon, a farmer; Sumner S. Sheldon, a farmer; Florence H. Sheldon Carrick (wife of The Reverend A. R. Carrick), and Julia Sheldon (Mrs. W. J.) Evans, who in 1941 was the only one living.

On June 27, 1853, to repeat, Dr. Sheldon arrived with his family at Wild Cat

Bluff or Brownsville, Houston County, where he soon bought 160 acres of land for $600. As there were only four families in the settlement, his medical skill was not demanded immediately to any great extent, but his legal knowledge soon was useful, for within a year he was presiding as judge at the first court held at Brownsville. And obviously he entered into community life, because on July 4, when a great barbecue was to be given at Brownsville in celebration of the day, Dr. Sheldon was one of the committee in charge of the arrangements. The following year, on November 9, he moved with his family to Mound Prairie Township and there, near the village of Mound Prairie, he established a large farm, the working of which in later years he turned over to his sons. It was characteristic of him throughout his career that he combined with his professional pursuits the occupation of farming.

Dr. Sheldon's broad professional training made him a most valuable citizen in the pioneer region. As noted, he was elected the first judge of probate in Brownsville, in 1854; in 1860 and again in 1862 he was elected court commissioner. Early convinced of the necessity of establishing settlements, he surveyed, platted and named the village of San Jacinto, secured for it a post office and acted as the first postmaster, and in the same period of the fifties he platted the village of Sheldon, in Sheldon Township. In 1859 he was one of the second board of supervisors of Houston County and, when the change was made from the system of government by supervisors to that of commissioners, in 1860, he was again a member of the county board. In 1863 he served as county coroner.

An able physician and surgeon, Dr. Sheldon often was called in consultation by other physicians in difficult cases. In his diaries, mentioned previously, he wrote of Drs. Train, Fox, Gates, White, Cowles, O'Connor, Bowen and Pope. An untiring student, a cultured and well-educated man, he possessed a good medical and general library, at one time the finest in the county. In the years in which the question was an issue, he was a vigorous opponent of Negro slavery.

On the farm in Mound Prairie Township, serving the community in many capacities, Dr. Sheldon lived until 1880, when death resulted from a stroke. Well-deserved tribute was paid him by all who knew him in recognition of his ability, his ambition and public spirit, the multiple civic and professional services he rendered in spite of his handicap of frail health, and his unfailing consideration of the sick and distressed. Mrs. Sheldon survived her husband many years and died in 1908, having shared with him the honor and esteem of the community.

Dr. Sheldon left many diaries, books, letters and instruments of historical interest and value which at the time these notes were written were in the possession of his grandson, Sumner S. Sheldon, Jr., of Houston.

Rolland Fuller Sheldon, who was to combine the offices of physician and clergyman, was born at Grafton, Vermont, in 1825, the son of Abraham Curtis Sheldon, who was a lineal descendant of Isaac Sheldon.

Isaac Sheldon, around 1640, emigrated from Derbyshire, England, to New England, America, and was one of the founders of Windsor, Connecticut, and Northampton, Massachusetts. To him and his wife were born fourteen children, nearly all of whom married and left children, who dispersed into various parts of the country, so that the Sheldon stock has been widely scattered. Of this family of fourteen, Abraham C. Sheldon was graduated from Yale University in 1812, taught in Middlebury Academy, Vermont, and about 1831

removed from Vermont to Portage, New York. When he was killed, in February, 1834, in felling a tree on his own land, he left his wife and seven small children to face pioneer existence in the great forest region of western New York. The children held together under the influence of their intelligent and able mother, won through to education and useful lives.

Rolland Fuller Sheldon became a master in many lines of work. He was carpenter and blacksmith, musician and music teacher, and in these and various other capacities he earned his way to the degree of Bachelor of Arts at the Baptist University of Rochester, New York, which was then a small institution of less than 300 students. Graduated in the class of 1856, he stayed on in Rochester through 1857 to study theology and medicine in preparation for becoming a foreign missionary. The university did not offer a medical course and Mr. Sheldon studied and practiced medicine with one of the leading physicians of the town. In 1858, therefore, when The Reverend Mr. Sheldon and his bride, Mary Adel Sheldon, arrived at the village of Sheldon, Sheldon Township, Houston County, Minnesota, as home missionaries, (having given up the idea of foreign service) of the Baptist Church, this young physican-clergyman was the possessor of as thorough preparation for the practice of medicine as were many of his solely medical contemporaries.

The village of Sheldon, which Dr. Sheldon made his headquarters, had been founded in 1854 by one of his cousins, Julius C. Sheldon, of Suffield, Connecticut, and platted by Dr. Giles J. Sheldon, to whom he is said to have been distantly related. Beautifully and well situated though it was, accessible by valleys in all directions, the settlement did not become the county seat, as was hoped by its founder, but became one of the disappointed hamlets left to one side by the railroad in 1866. Letters and documents in the possession of Dr. Sheldon's son show that this pioneer physician and clergyman traveled with horse and buggy not only in his own township and county but also over much of southeastern Minnesota, carrying his ministry of grace and healing from the Mississippi River as far west as Carimona and Cherry Valley, in Fillmore County, and as far north as Chatfield, in Fillmore and Olmsted Counties, and Winona, in Winona County. He was a leader in the settlement and organization of this entire territory. In 1860 he was an ardent advocate of Abraham Lincoln for President and, when the Civil War broke out, he was active in raising money and recruiting men for defense of the Union; although because of his dependents, a wife and two small children, he did not enlist; he was unremitting in his patriotic efforts. His younger brother, Abraham Sheldon, of Cherry Grove, enlisted in the army for defense of New Ulm and Fort Ridgely during the uprising of the Sioux Indians in 1862.

When an epidemic of black measles in virulent form broke out in the Norwegian settlements of Black Hammer Township in 1863, Dr. Sheldon was tireless in the campaign to combat the lethal disease, carrying medicine and counsel to afflicted homes, as was customary in the office of the physician in those days. Repeatedly exposed to the disease and overweary, he was already sickening when one night he was caught in a violent rainstorm as he drove home from an errand of mercy to Black Hammer. The next day he came down with the measles and on April 3, 1863, after a few days of raging fever and delirium, he died. He was buried in the old Pioneer Cemetery in southwestern Caledonia in the lot adjoining that of the Trask family, who were among his closest friends.

Dr. Sheldon kept always a large supply of medicines in an old-fashioned

red chest that he and his wife had brought from the East, and after his death Mrs. Sheldon dispensed these drugs as he had taught her to do. In the succeeding years, during the Civil War and later, Mary Adel Sheldon, a well-educated woman, taught school in Caledonia, Yucatan, Winnebago Valley, Looney Valley, Sheldon and Houston, all in Houston County. There remains record in old files of Caledonia newspapers of a great patriotic parade, during the war, of all the children of the Caledonia schools, led by Mrs. Sheldon.

In 1943 there were living, among other relatives of Dr. Rolland Fuller Sheldon, his son, Addison E. Sheldon, Superintendent-Secretary of the Nebraska State Historical Society, at Lincoln; his grandson, Philip Sheldon, and his great grandson, Keith Sheldon, both, also, of Nebraska.

Trond Neilson Stabo, born in Norway in 1869, was graduated in 1894 from the Fredericiana University (Kongelige Frederiks Universitet) at Christiania (Oslo). Not long afterward he migrated to the United States and came to southern Minnesota, and on October 8, 1895, he was licensed to practice in the state. On the following October 17 the certificate was recorded in Houston County.

A resident of Spring Grove, Dr. Stabo was a well-known practitioner throughout the county and in contiguous counties. It has been recalled that he was probably one of the first in the county to perform surgical operations for appendicitis, and that he owned one of the first automobiles in the region of Spring Grove and Caledonia. In those days, according to an early citizen of Caledonia, there were no real automobile mechanics and in the smaller towns when anything went wrong with an automobile, the blacksmith worked on it. Dr. Stabo, on one occasion having difficulty with a new car which was giving out clouds of black smoke, was insistent that the magneto should not be touched because, he said, it was sensitive, as stated in the directions that had come with the car. When the difficulty had been corrected as a result of tactful adjustment by the mechanic, who it happened was not a blacksmith, and the doctor was able to continue his rounds with a perfect running car, he returned and paid generously for the correction.

Dr. Stabo was a member of the Houston-Fillmore County Medical Society, the Minnesota State Medical Association and the American Medical Association. In 1907 he moved to Decorah, Iowa, where, in 1943, he was still practicing.

Of **E. D. Stewart,** said to have practiced in Houston County, information has not been available.

Gilbert Thomas was listed in a commercial directory of the late seventies as a physician in the village of Yucatan.

Isaac Whittington Timmons, born on October 11, 1833, on a farm near Chillicothe, Ross County, Ohio, was to become one of the well-known pioneer physicians of southern Minnesota, in Houston, Fillmore and Winona Counties. He was the son of Thomas Whittington Timmons, whose father had come from England with Lord Baltimore and had settled in Baltimore (at first called St. Marysland), Maryland. Isaac Timmon's mother was Hetty Draper Timmons, educated in Philadelphia, the daughter of a Quaker family of Delaware which included a son who became a physician. Thomas Timmons

married a second time, a younger sister of Hetty Draper, and of the marriage there was one daughter, Hester.

Isaac Timmons received his early education in the private and public schools of Chillicothe until, in 1851, when he was eighteen years old, he took up his residence in Matamore, Illinois. About three years later in Matamore it happened that he was obliged to go into court for settlement of a dispute, and his attorney was none other than Abraham Lincoln. The difficulty was occasioned by the fact that Mr. Timmons had been elected to the office of town clerk two days before he was twenty-one years of age. He was notified, on his birthday, of his election and on the following day, after being duly sworn in, he gave bonds. Mr. Lincoln, when the legality of his assumption of office was contested, offered his services as defending attorney and brought Mr. Timmons through a victor. At that time Mr. Lincoln remarked that the boy had begun office-seeking early in life and added, when told that friends had been responsible for the candidacy, "It is well to have a good lot of friends when you run; I have met defeat once or twice because I didn't have quite enough."

In the middle fifties Isaac Timmons studied medicine under the preceptorship of one of his brothers, who was a physician, but the instruction was interrupted when in the late fifties he moved to Cairo, Illinois, to engage in the mercantile business with another brother. In Cairo, at the southern tip of Illinois, separated from slave-holding territory only by the Ohio and the Mississippi Rivers, he served as a member of the common council in 1860 and 1861 at the time, just before and after the outbreak of the Civil War, when excitement ran high along the border. By 1862 he had settled in Centralia, Illinois, where for the next eight years he was employed in the service of the government as deputy collector of internal revenue for some eleven counties. During those years he was associated with Robert Noleman, uncle of William Jennings Bryan, and grew to know Judge Bryan, father of "Little William," who then was four or five years of age.

During his residence at Centralia, Isaac Timmons studied law and served as police justice, but law did not appeal to him and he returned to the study of medicine. In 1870, after a course of study of three years at the Homeopathic Medical College of St. Louis, Missouri, he was graduated with the degree of Doctor of Medicine, and in the same year he entered the state of Minnesota to practice medicine for a short time in the little village of Money Creek, in Money Creek Township, Houston County.

For the next twelve years he was, as his daughter has described him, "the proverbial rolling stone." In the early seventies he went to Winona, where he practiced in partnership with Dr. T. Pierce. The pioneer river town disappointed him, however; the levee was a sand and gravel bank and the streets were full of mud and holes. A few plank walks were to be found, but a "general untidy state of affairs existed." Years later he was very proud of Winona, and justly, for he had played a part in building up the beautiful city.

After two years in Winona, in 1877, Dr. Timmons returned to Houston County, this time to the village of Houston, where he stayed until 1881. Besides carrying on a general medical practice he served as justice and as county physician. In 1881 he went to North La Crosse, Wisconsin, but on learning that Chatfield, Fillmore County, Minnesota, was in need of a physician, he settled there for a year and a half. In the early spring of 1884 he returned to Winona where he built up a successful practice and remained there until his death thirty years later. After 1887 he practiced under the Affidavit Ruling.

Isaac W. Timmons was married, in 1857, to Ursula E. Robinson, of Chillicothe, Illinois. Dr. and Mrs. Timmons had six children, Virginia, Jessie, Kate, Lida, Luetta and John W. Timmons. Mrs. Timmons exerted a gentle and understanding influence on her husband's career. In later years the doctor always referred to their life together as "a continual Indian Summer." In 1907, when Dr. and Mrs. Timmons celebrated their golden wedding anniversary in Winona, all but two of the children were living and present. In 1941 only one, Luetta (Mrs. C. A. Peiffer, of Winona) was living. The son, John Whittington Timmons, was for many years, until his death in 1918, a teller of the First National Bank of St. Paul. Kate died in 1937 at Long Beach, California, at the age of eighty-two years. Lida, the wife of C. F. Spencer, who was editor of the *Willmar Journal* for many years, died at Willmar, Minnesota, in 1941. Virginia ("Jennie"), who did not marry, taught school for many years; she died in Kansas City previous to 1907. Jessie, who was married, also died before 1907.

Dr. Timmons was a great reader and student who always took a keen interest in politics. He used neither alcoholic liquor nor tobacco, was a member and supporter of the Methodist Church, and was generous in giving his professional services to those who were unable to pay.

On October 9, 1912, two days before his seventh-ninth birthday, Dr. Timmons died from a stroke.

Hamilton B. Train was born in St. Lawrence County, New York, on April 26, 1825. By 1847 he was in Wisconsin, studying medicine and, after he had settled in Fond du Lac, preaching the gospel. Because of ill health he was obliged to give up the ministry and shortly thereafter he enrolled at Appleton University for a period of medical study. After his return to Fond du Lac he was married to Angelina Clark, on March 27, 1850. Dr. and Mrs. Train had four children, of whom two were living in 1882 (the date of the last published comment available).

In 1856 Dr. Train, a physician of the regular school, came to Minnesota and in the following year to Hokah, in Hokah Township, Houston County, where Dr. Charles Jenks was the only practitioner of medicine, having arrived a short time previously in 1856. Hokah was then at the beginning of a period of mounting prosperity which continued until 1872 when, with the reduction in status of the railroad shops and the corresponding reduction in population and business enterprises of all types in the community, a permanent decline began. Dr. Train, well established, early had built up an excellent practice and he made Hokah his permanent home. In 1880 and 1881 he was serving as health officer for the township during an outbreak of smallpox in the locality. After the "Diploma Law" of 1883 came into effect he practiced under a state exemption certificate. He died in Hokah on July 28, 1891, thirty-four years after his arrival in the village. Mrs. Train continued to make her home in the village after her husband's death.

Dirk Van Krevelen was born in Holland in 1828. His preliminary education and his academic collegiate training he received in his native country and his medical education, at the universities of Germany. It has been said that he arrived in Baltimore, Maryland, in 1854 and that soon afterward, accompanied by his family, he went to Valparaiso, Chile, on appointment as superintendent of a marine hospital there. During a political and religious uprising in Chile in 1858 and 1859 he, together with his wife and children, was rescued from

the city by a sloop-of-war, but only after they had experienced severe difficulties and hardships; ultimately they returned to Baltimore. During the Civil War Dr. Van Krevelen served as surgeon on the field and in hospitals. After the war he spent a year in Europe in medical study and in renewing old friendships, and on his return to the United States, settled in Grand Rapids, Michigan, and there practiced medicine.

In 1849 Dr. Van Krevelen was married for the first time. His wife died in 1867 and in 1873 he was married to Gertrude Edons, like himself, a native of Holland.

By 1879, at the latest, and perhaps a year or two earlier, he was in Belle Plaine, Minnesota, and it has been said that he moved there from Hokah, in Houston County. An able physician and a well-liked, genial man in Scott County, as elsewhere, he won friends and confidence and had a good practice. In 1880 he served as county coroner. By 1888 he had settled in Carver County, near New Germany, where he bought a general store, which he proceeded to expand. Not of mercantile taste, however, he soon sold out and next chose Waconia as his field of professional endeavor. In 1897 he moved with his family from Waconia to Silver Lake, in McLeod County. In the official register of 1883-1890 of licensed physicians in Minnesota he was listed, without specified residence, as the holder of an exemption certificate.

M. J. Veiling (Velling?) probably was one of the first few physicians, certainly he was among the earliest group of physicians, in Caledonia and in the county. It appears from early records that he was the second coroner, in 1857, of Houston County and that he was succeeded in the office some time prior to 1868 by Drs. Harvey B. Laflin and Timothy A. Pope; Dr. Veiling served a second term in 1862. It is hoped that more will be learned about him, perhaps through reports on early physicians in different parts of the state.

Charles H. Wagner, possibly the Dr. Charles H. Wagner who was more closely identified with Fillmore County in later years, was listed in a business gazetteer of 1880 and 1881 as resident in Money Creek, Houston County.

S. C. White was mentioned by Dr. Giles J. Sheldon in his diaries as a contemporary physician in Houston County, obviously some time during the period between 1853 and 1880. It is known that in 1881 and 1882 Dr. White was serving as health officer in the village of Hokah at the same time that Dr. H. B. Train was health officer for Hokah Township and that Dr. W. W. Holden was health officer for Union Township. Not to confuse the issue, but rather to note the possibility of a misprint in initials of the same man, it should be stated that in the autumn of 1871 a Dr. A. C. White, physician and surgeon, lately of Muskegon, Michigan, opened an office in Preston, Fillmore County. Changes of residence from county to county were common among physicians in those years and it is possible that Dr. White subsequently settled in Houston County.

(To be continued in the March issue)

President's Letter

Two items of economic development in medicine, which have recently come to the attention of the Council and officers of the Association, are of such vital concern to the entire membership that comment upon them now seems to be an urgent necessity.

The first of these is the prediction that the Wagner-Murray-Dingell Bill may pass at this session of Congress. To accomplish this, the authors of the bill have deleted from it all clauses involving the appropriation of funds. This has been arranged to ensure consideration of the bill by a committee friendly to it. Favorable action on a bill by the committee to which it is referred is followed by its introduction to the entire Senate with the recommendation by the committee that it be passed.

These facts mean that any opposition to the bill will have two opportunities to defeat it. The first opportunity will be on the floor of the Senate where it is subject to debate by the entire Senate prior to its being voted upon. If the opposition to the bill can summon enough strength on the floor of the Senate, the act can be defeated by general vote of that body. In spite of a press poll of the Senate indicating the bill's defeat, some predictions are that this is not likely. The second opportunity to prevent its full enactment will be in defeating the appropriation bill which will have to be passed later to provide the funds necessary to carry out its provisions. At present, the latter course appears a more likely point of obstruction than defeat of the initial bill on the Senate floor.

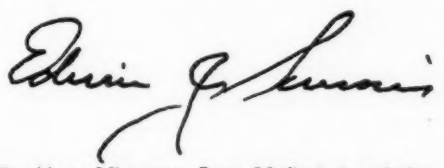
It should be borne in mind that under the present system of medicine in the United States, health, mortality and longevity are better than those of any country in the world. The issue in the Wagner-Murray-Dingell Bill is supposedly the reduction in cost of medical care. In every instance where compulsory Health Insurance has been inaugurated to reduce the cost of medical care, quality of medical care has been sacrificed. So, in the present instance of the Wagner-Murray-Dingell Bill, the important fact is that not only is the American System of Medicine at stake, but, of far greater importance, the actual health, lives and longevity of the people of this country are being voted upon in the halls of Congress.

The second matter of economic importance to the profession is the reorganization of veteran medical care. Veterans Hospital facilities in Minneapolis are being integrated with those of the Medical School and University Hospital. Veterans Administration branch offices are being established throughout Minnesota. Medical care of the veteran in his home community may be provided by the Veterans Facility in accordance with a plan now being arranged by a committee of the Minnesota State Medical Association.

At the Veterans Hospital in Minneapolis, all physicians will be enrolled in residencies or fellowships with University Hospital and Medical School faculty members serving as consultants or senior consultants. All gradations of physician appointments will be co-ordinated with postgraduate medical education so that each staff member may, under the guidance of proper authorities, ultimately become a diplomate of some specialty board.

In order to facilitate medical care of the veteran in his home locality, branch and administrative Veterans Facility offices are to be established in a number of the cities scattered throughout the state. Through these offices, physicians called in attendance of a veteran in his home can secure, by telephone, permission to provide medical care. It is expected that both administrative and medical personnel will be found in the branch offices.

For the preparation of details of a program to render medical care to the veteran in his home community, a committee of physicians has been appointed for the Minnesota State Medical Association. The committee consists of physicians who served in this or the first World War. All sections of the state and all branches of the military service are represented on the committee. Fee schedules for such veteran medical care are being promulgated. Service connected and nonservice connected disabilities are receiving consideration. As soon as a comprehensive, detailed plan is completed, it is to be sent to Washington for approval. All of these developments are in the process of completion. Details will be provided as they are available.



President, Minnesota State Medical Association

Editorial

CARL B. DRAKE, M.D., *Editor;* GEORGE EARL, M.D., HENRY L. ULRICH, M.D., *Associate Editors*

IMPROVED VETERANS' FACILITIES

THE veteran of World War II is going to be provided with the best possible hospital care. For some time considerable criticism has been voiced of the quality of care being furnished by the Veterans' Facilities.

Recently the President signed a bill which provided for Medical and Surgical Departments in the Veterans Administration. More than that, arrangements have already been made for the employment of specialists on certain medical school faculties on a half-time basis to care for these veterans. These new appointments have already been made in Minnesota by the Dean of the Medical School who with a medical faculty committee will supervise their activities. Civil Service will no longer designate appointments. It is understood that medical personnel in the Veterans' Facilities who hold their appointments under the Civil Service will be retained unless there is good reason to dispense with their services. Even though Civil Service appointments eliminate the undesirability of political appointment, it has long been recognized that the difficulty in dispensing with Civil Service appointees is at times a handicap.

Residents and interns at these Veterans' Hospitals will be appointed by the medical school which will have direct supervision of their activities. The value of residencies and internships in these hospitals to recent graduates and returned medical officers is likely to be secondary to none, and the new setup helps solve a pressing need. This presents a happy solution of a difficult problem.

Steps are being taken for a better solution of the care of the returned veteran who is in need of medical care for disabilities sustained in service but who does not perhaps need hospital care, who lives distant from a Veterans' Hospital or for whom perhaps there is no room at present in a Veterans' Hospital. There will undoubtedly be two or three times the demand for medical care on the part of veterans of World War II as there were following World War I. How can this best be provided? It is the idea of Major General Hawley, chief medical director of the Veterans Administration, that the medical profession of each state present their own plans for his consideration.

The medical profession of Michigan has already submitted its plan whereby the members of the Medical Services, Inc., of Michigan will provide medical care for veterans outside of the Veterans' Hospital on a fee basis acceptable to the members of the Michigan State Medical Association. Steps are being taken to provide hospital care also through the Blue Cross hospital organization in Michigan.

Personally, we can see no need for any other organization than the State Medical Association when it comes to considering ways and means for providing this medical service for Veterans of World War II who do not require care in a Veterans' Hospital. All that would be necessary would be an agreement on fees for certain services and an indication of willingness on the part of the member to fill out certain forms. We have a committee of our State Medical Association working on the project now.

Following World War I methods adopted for providing for disabilities of veterans were often extravagant in time and money in that veterans were required to travel long distances to receive medical aid. Utilization of the services of the veterans own medical advisor would eliminate this unnecessary expense. The local physician can care for veterans suffering from certain disabilities in the home or local hospital, without necessitating care in a Veterans' Hospital.

SCHEMM TREATMENT OF EDEMA

IN April, 1942, Dr. F. W. Schemm of Great Falls, Montana, read a paper[1] before the annual meeting of the American College of Physicians, in Saint Paul, on the value of a high fluid, a low salt and sodium intake and an acid ash diet in patients suffering from edema from any cause and especially from heart disease. Before this paper was published another[2] by the same author appeared. In spite of the array of cases scientifi-

cally studied and the most favorable results reported, the profession has been slow in accepting his contribution.

In spite of numerous authoritative statements which have appeared in the literature during the past hundred and fifty years on the value of water as a diuretic, the medical profession today is rather generally disposed to restrict fluids in edema from any cause. This is apparently wrong and not only adds to the discomfort of the patient but may even result in a dehydration of tissue cells and a lessening in the elimination through the kidneys of the sodium chloride and sodium bicarbonate contained in the edema fluid.

Schemm argues the logic of reducing to a minimum the sodium chloride and soda intake in the diet and prescribing an acid ash diet to aid the neutralization and elimination of the sodium bicarbonate in the edema fluid, although the acid normally formed in the tissue will be sufficient in many cases. He sometimes adds acid in the diet in the form of dilute HCL acid or ammonium chloride to assist this process.

Foods that produce an acid ash are meat, fowl, fish, eggs, cereals (except corn) prunes, plums and cranberries. All other foods yield an alkaline ash.

His treatment consists of forcing liquids, particularly water, and giving a neutral diet consisting of low salt, low sodium and an acid ash diet. No food containing salt and no soda are allowed. Small amounts of milk and certain vegetables and fruit are allowed in addition to the acid ash foods.

Digitalis, when indicated, should of course be continued. Murcurial diuretics may also be used although they are seldom needed in obtaining diuresis.

Schemm's detailed reports of his success are most convincing, and trial of his method has confirmed his observations.

1. Schemm, F. R.: Am. Int. Med., 17:952, (Dec.) 1942.
2. Schemm, F. R.: A high fluid intake in the management of edema. Am. Int. Med., 21:936, (Dec.) 1944.

DRUGGISTS IN DISTRESS

PROVIDING the druggist with prescriptions for narcotics before the prescription is filled oftentimes causes delay or inconvenience for the physician or patient or both. When the prescription is for a simple cough syrup containing codein which is needed to help the patient through the night, the insistence on the part of the druggist that he have the written prescription before he delivers the medicine may seem unreasonable to the physician.

Nevertheless, the Bureau of Narcotics is responsible for the proper dispensing of narcotics and has established the following regulations:

Article 172—Bureau of Narcotics Regulation No. 5

Telephone Orders—The furnishing of narcotics pursuant to telephone advice of practitioners is prohibitive, whether prescriptions covering such orders are subsequently received or not, except that in an emergency a druggist may deliver narcotics through his employe or responsible agent pursuant to a telephone order, provided the employe or agent is supplied with a properly prepared prescription before delivery is made, which prescription shall be turned over to the druggist and filed by him as required by law.

There is reported to be a scarcity in the illicit traffic of narcotics. The addict will usually go to any length to satisfy his craving. It is not unusual for him to call the druggist, posing as a physician and ordering a narcotic. There is good reason, therefore, for insistence on the part of the Bureau of Narcotics that the druggist have the physician's prescription in his hands before he furnishes the narcotic. Too often the physician becomes irritated with the druggist who seems to be lacking in co-operating with his patron, the physician. The Narcotics Bureau, however, is clamping down on druggists who fill narcotic prescriptions over the telephone. Under the circumstances, one cannot blame the druggist if he is meticulous in observing the letter of the regulation.

PENICILLIN ACTION NOT NEW

AN interesting communication from Dr. Russell F. Maddren appeared under Correspondence in the *Journal of the American Medical Association* for January 19, 1946. He refers to a communication from the physicist, John Tyndall, to the English Royal Society, which was published in the Philosophical Transactions in 1876.

Tyndall reported the clearing of exposed tubes containing bacteria and his inability to find any active life in the bacteria in tubes contaminated with surface growths of penicillium glaucum.

Thus the fact that the mold Penicillium inhibits the growth of bacteria was recognized several years ago, but the therapeutic significance of this fact was not recognized until recently. Truly, knowledge comes but wisdom lingers.

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

NATIONAL HEALTH CONGRESS PROPOSED

A National Health Congress, as an answer to the threat of government control of the health professions, was the subject of a set of resolutions presented before the annual meeting last month in Chicago of the AMA House of Delegates.

According to Dr. E. S. Hamilton, Illinois, who introduced the resolutions, such a congress is urgently needed to prevent regimentation of the medical profession, to preserve and extend health services for this nation's citizens and to provide a co-ordinating body for all agencies in the health field.

These resolutions represent the first step in making the National Health Congress more than just an idea. It was proposed:

1. That the House of Delegates "recognize the need for a National Health Congress, representative of the medical, dental, hospital, nursing, pharmaceutical and allied professions, and that it approve the creation of such a co-ordinating body."
2. That the House of Delegates instruct the Council on Medical Service and Public Relations to take the initiative and "bring together interested groups to organize and incorporate *immediately* a National Health Congress."

The resolutions were referred to the Reference Committee on Legislation and Public Relations, which reported the resolutions to the floor where they were subsequently passed.

What Is the National Health Congress?

The idea of a National Health Congress has been in the making for a long time. Physicians will be hearing more and more about it in regard to the controversy between those favoring federal control of health services and those of the health professions who contend that this nation's health services must and shall remain in free hands.

Dr. A. S. Brunk, former president of the Michigan State Medical association, was instrumental in formulating a concrete plan for the setting up of a National Health Congress. Collaborating with John R. Mannix, director, Plan for Hospital Care in Chicago, and John F. Hunt, vice president of Foote, Cone and Belding, Chicago, he presented an organizational outline in the October, 1945, issue of "The Modern Hospital." It is this outline that will be described here.

Among the several objectives of a National Health Congress presented in this report are: (1) keeping health protection for the American people on a voluntary basis; (2) insuring the public complete protection regardless of financial status or location; (3) co-ordinating all health agencies and activities; (4) providing a plan for voluntary prepayment of health protection at a cost equal to or lower than any national compulsory health insurance plan; (5) educating the public in health matters with the object of improving national health standards and interesting the public in taking advantage more and more of the facilities for health improvement; and (6) providing incentives for persons engaged in all phases of health work.

How Would It Be Organized?

According to the proposal, the National Health Congress would be patterned after our national law-making body, so that it would not only serve as a forum for free and open discussions on health matters, but would also be endowed with the power to act for the nation's voluntary health forces, which would create it.

As for the membership, it is proposed that the Congress contain one or more representatives

of the hospital, medical, dental, nursing and pharmaceutical associations of each state as well as industrial, labor and agricultural associations from every state and representatives from the senate, the house of representatives and the United States Public Health service.

The National Health Congress would concern itself with all measures affecting health care at the national level. The Congress would cover any plan for prepayment of health care by an individual or family unit, and it would recommend to federal and state governments how they might participate in health activities, provide for research and extend benefits to public charges. To carry out the directives of the Congress, Dr. Brunk and his colleagues propose that a National Health Administration be set up under the direct control of the parent organization.

Must Have Permanent Quarters

The planners of the National Health Congress recommend that the Congress have permanent headquarters in some city equally accessible to all members as well as capable of providing facilities for carrying on the administrative functions of the Congress. The body would convene once a year, at least, to consider problems of extension and improvement of the nation's health care; and, since it would be restricted to matters of health alone, it is not anticipated that members will find that serving with the Congress interferes too much with private affairs. Above all, the Congress must have the *power to act* so that the greatest amount of work can be accomplished in the shortest possible time. To expedite the work of the Congress, a system of committees would be set up to study individual proposals before they are voted upon. In this and in other ways, it would borrow the best aspects of the national legislative congress. This, then, is one plan offered as a counter-proposal to the compulsory health insurance plan outlined in the Murray-Wagner-Dingell Bill.

Organization of a Health Congress Forthcoming

From the action recently taken by the AMA, it is evident that machinery will be set in motion shortly to create a National Health Congress, perhaps one similar to that proposed by Dr. Brunk and his associates. In a news letter sent out January 15 by the Council on Medical Service and Public Relations to officials of state associations, the Council reported that the formation of a National Health Congress is one of the very important tasks it is commissioned by the House of Delegates to carry out during 1946, another being the development of a specific national health program. In the words of the Reference committee which presented the resolutions, a National Health Congress "will undertake to bring to all the people of this nation the complete benefits of modern medical-dental science and the finest hospital facilities, will arrange for budgeting the cost of such services at monthly rates within the financial means of all Americans through voluntary, non-profit health plans and will work speedily for the institution of such voluntary non-profit health plans in those places or localities where they do not now exist."

Citing the need for immediate establishment of such an organization, the resolutions stated further: "While each of the groups in the health field has its own organization, working independently for the preservation of the American system of health care, an over-all body or council is necessary to integrate the necessary collective thinking and activity of all organizations or units." For that reason a serious and immediate need exists to develop a working liaison or congress of all agencies in the health field to secure the most effective results of action and public education.

PUBLIC HEALTH PROFESSORSHIP CREATED AT "U"

The University of Minnesota was recently on receiving end of a gift of $150,000, which will finance the University's first permanently endowed professorship. The donor of the gift was the Mayo Properties association.

Salary for the post, to be known as the Mayo Professorship in Public Health, is to be not more than $10,000 annually, and will probably be filled by July 1, according to University authorities.

The University has one other named professorship, the George Chase Christian Professorship in Cancer Research. This chair is not permanent, however, although it is still being maintained by gifts received at the time of the liquidation of the Citizens' Aid society.

The school of public health, which will contain the new post, was created within the medical school two years ago from what was formerly the department of preventive medicine and public

health. It will serve as one of this country's six regional training centers, where doctors, engineers and nurses in public health, studying at the graduate level, can become specialists.

VET HOSPITAL EXPANDS MEDICAL STAFF

The recent addition of 146 physicians, many of them nationally known men in their fields, to the Fort Snelling Veterans hospital staff is expected to make the hospital one of the nation's leading veterans' medical centers. A joint announcement of these additions to the staff and plans to have the University of Minnesota medical school take over most of the responsibility for medical services at the hospital was made by Dr. Harold S. Diehl, dean of medical sciences at the University; Carl D. Hibbard, manager of the Minneapolis veterans administration facility; and Dr. Harry E. Bank, chief medical officer at the veterans hospital.

Broad Plan Outlined

A broad program of improvement of facilities of the veterans hospital has been outlined which will add to the staff 35 men to hold the rank of senior consultant, 43 to serve as consultants and 68 resident physicians or fellows. The program provides:

1. Direct supervision of all appointments of doctors to the hospital staff by the dean of the University medical school so as to raise the hospital standards to the level of those of the medical school;

2. The addition to the staff of specialists from all parts of the country to be supervised by a University committee, headed by Dean Diehl;

3. The establishment of a fellowship system and the appointment of 68 "highly selected" University graduate doctors under the system to work full-time in the hospital to receive specialists' ratings; and

4. The appointment of physicians by merit and ability rather than on the basis of civil service status, veterans to be given preference when possible.

Aim to Modernize System

It is reported that the Veterans hospital at Fort Snelling is the second in this country to modernize its system. The purpose of the modernization, according to veterans administration authorities is "to insure that veterans will obtain the same up-to-date medical care and treatment that they would obtain in hospitals attached to the nation's leading medical schools and centers."

Dr. Diehl puts it this way: "This plan will give the veterans the kind of treatment I'd want my own son to have."

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul, Minnesota

Julian F. DuBois, M.D., Secretary

Minneapolis Osteopath Pleads Guilty to Criminal Abortion

Re State of Minnesota vs. Madison J. Hampton

On January 9, 1946, Madison J. Hampton, fifty-nine years of age, and licensed to practice osteopathy in the State of Minnesota, entered a plea of guilty in the District Court of Hennepin County, to an information charging him with the crime of abortion. Hampton, who for the past year had maintained an office at 1406 W. Lake Street, Minneapolis, and whose office for fifteen years previous to that time was in the Masonic Temple Building, Minneapolis, was sentenced by the Hon. Arthur W. Selover, Judge of the Hennepin County District Court, to a term of one year in the Minneapolis Workhouse. Upon the recommendation of Mr. Otto Morck, First Assistant County Attorney of Hennepin County, and F. Manley Brist, attorney for the Minnesota State Board of Medical Examiners, Judge Selover stayed the sentence upon condition that Hampton close his office and refrain in every manner, from practicing osteopathy and violating the laws of the State of Minnesota.

Hampton was arrested on December 13, 1945, following an investigation by the Minneapolis Police Department and the Minnesota State Board of Medical Examiners into Hampton's activities. On December 4, 1945, a thirty-four-year-old married Minneapolis woman was admitted to General Hospital, suffering from peritonitis following a criminal abortion. The woman gave a statement naming Hampton as the person who performed the abortion upon her. Hampton, at the time of his arrest, gave a signed statement in which he admitted having performed the abortion by means of "a blunt wire silver instrument" which he used to curette the patient. Hampton also admitted in his signed statement that he was paid $65.00 for the criminal abortion, $35.00 of which he returned to the patient when it became necessary for her to be hospitalized. Hampton also admitted that he had been performing abortions over a period of the past ten years. Hampton stated that he graduated from the Des Moines Still College of Osteopathy and that he had been practicing in Minneapolis since 1920.

Minneapolis Surgical Society

Stated Meeting Held November 1, 1945

The President, Robert F. McGandy, M.D., in the Chair

Dr. Felipe Torres of Mexico, by invitation, spoke on "The Teaching of Medicine in Mexico and Its Evolution."

EXPERIENCES IN AN EVACUATION HOSPITAL IN ETO

R. E. HULTKRANS, M.D.
Minneapolis, Minnesota

The mechanical operation of an Evacuation Hospital of 400-bed capacity was discussed.

The personnel consisted of thirty-nine officers, forty nurses, and 217 enlisted men. During all of the campaign in France and Germany, this unit functioned in tents except for three months during the winter.

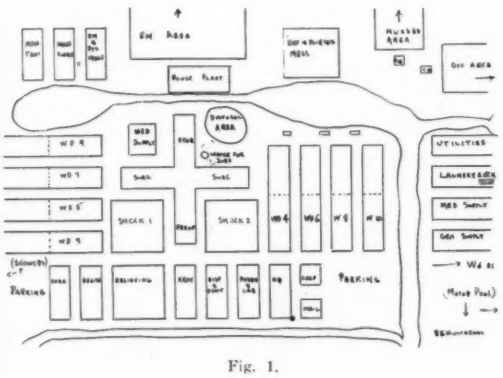


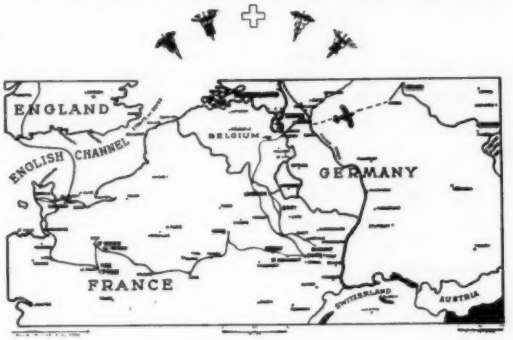
Fig. 1.

The hospital was transported from one station to another in trucks except at the time of the Rhineland campaign when the entire unit including equipment and personnel was flown in C-47 planes a distance of about 200 miles. The hospital was in operation at thirteen different stations in France and Germany.

Figure 1 shows an average setup of the hospital. The tents used were 40 by 16 feet. The large squares represent two such tents tied together at the sides and set up as a single tent. The double oblongs represent two tents tied end to end.

Figure 2 is a map of the intinerary of the hospital and shows the areas at which the hospital was in operation.

This type of unit proved highly efficient; 20,738 casualties were treated up to V-E Day with a mortality rate of slightly less than 3 per cent. All types of cases of both a medical and surgical nature were handled.



20 July 1944—9 Aug. 1944	Carentan, France	1,105
10 Aug. 1944—17 Aug. 1944	Yvre-Leveque, France	566
17 Aug. 1944—27 Aug. 1944	St. Gervais-duPerron, France	963
29 Aug. 1944—12 Sept. 1944	Normer, France	961
12 Sept. 1944—18 Sept. 1944	Germany, France	429
18 Sept. 1944—24 Sept. 1944	St. Remiremont, France	373
24 Sept. 1944—15 Oct. 1944	Chatel-sur-Moselle, France	1,869
15 Oct. 1944—21 Dec. 1944	Bricy, France	1,116
21 Dec. 1944—26 Jan. 1945	Longuyon, France	5,030
3 Feb. 1945—22 Feb. 1945	Baccarat, France	831
6 Mar. 1945—31 Mar. 1945	Bergheim, Germany	1,579
10 April 1945—20 April 1945	Gottingen, Germany	1,756
20 April 1945—V-E day	Zeulenroda, Germany	1,127
Total Admission		20,738

Fig. 2.

Discussion

DR. CHARLES ALING: I think after that excellent discussion by Doctor Hultkrans on Evacuation Hospitals, it is superfluous for me to add anything. I know you realize that no one can claim more experience in an Evacuation Hospital than he. His unit did see a tremendous amount of work. Our unit followed very much the same course, but were a little further north.

We found that we couldn't operate for a too long period of time, for after a definite length of time, one's efficiency would go down. We think we are unique in developing an eight-hour shift. We operated continuously for eight hours, except one-half hour for lunch. Each shift would alternate, reporting for duty every other eight hours. We found also that, by this shift, doctors and nurses had every other evening off. This would be early enough to obtain some recreation to help them forget their troubles. On a twelve-hour shift one would be on days for two weeks, for instance, necessitating the alternate shift being on nights for the same period.

CALCULATION OF DISABILITY IN THE EXTREMITIES

CARL O. RICE, M.D.
Minneapolis, Minnesota

In order to establish a method whereby disability can be standardized, it seems necessary to establish a few dicta by which to guide disability calculations so that the injured individual could be given the benefit of the doubt; whereby the compensation laws can be taken into consideration; and in which the insurance carrier can feel that he is not being required to pay excessively where a relatively minor disability occurred. These dicta are as follows:

1. In one hundred per cent industrial disability arrives somewhat short of 100 per cent uselessness. Most state industrial laws specify that loss of use of a member constitutes loss of that member. This seems reasonable for a member is of no industrial value if it cannot be used, even though it remains attached to the body.

2. Disability of a single joint is a relatively greater handicap to that member, when involved alone, than when involved with other joints. Therefore a single joint must receive a greater relative value in relation to the whole member than it receives when involved with one or more joints of that member. This is in harmony with the industrial law which specifies a greater total value for the loss of a thumb and all the fingers than for a hand.

3. Each joint of a member should be given a specific value in relation to the member as a whole. These relative values should indicate the disability to the member when the joint is completely rigid and unassociated with other disabilities.

4. Disability should be calculated upon objective measurable factors in order that some degree of constancy can be established. The degree of a motion is the most objective measurable factor. Additional estimates of minor value for disability may be added for other factors, not measurable, in order to allow for pain, loss of sensation, in-coördination, lack of power, et cetera. But if the serviceable range of motion is used as the measurable factor, then most of these other factors have been given due consideration. It has seemed to me that methods for culculating disability wherein pain, in-coördination, weakness, adaptability, et cetera, have been given unit values are too intangible to be much more than a good guess, accurate only in proportion to the experience of the physician who gives it.

5. The final percentage of disability in a member should be constant, no matter in what State it exists or in what occupation it occurs. The award for such disability may vary depending upon occupation or other factors, but that is a problem for the Industrial Commission to establish and not a problem for the physician. The loss of an index finger constitutes a loss of 100 per cent whether on a concert violinist or a ditch digger.

6. Disability cannot be measured within more accurate units than five or ten degrees. Therefore, the end result should not be calculated more accurately than in units of five. The end figure should be placed at the closed multiple of five. This I choose to call a disability unit.

It seems further necessary to determine the nature of the existing laws in order that disability evaluations may be compatible with the law.

The law indicates that compensation may be paid on the basis of a specified amount of money, for a specified number of weeks, for a specified loss of a member.

The law specifies these members: a thumb, a finger (index, middle, ring, and little); a hand, a hand and wrist, (or hand and forearm) an entire arm (upper extremity), a great toe, a toe other than the great toe (individual toes not specified), a foot, a foot and ankle, (foot and leg below the knee) a leg through the thigh, an entire leg (lower extremity).

Each disability estimation, no matter how small, must be expressed as a percentage value of one of these. The smallest member into which all of the disability can be included, is chosen as the one on which the disability must be calculated. If the disability lies within a finger, the calculation is based on a finger instead of the hand. If it lies within the hand it is based on the hand instead of the "hand and wrist" or "arm," et cetera.

It therefore becomes necessary to establish a relative value for the various portions of a member, in relation to a whole member, before it becomes possible to calculate disability to the whole of this member, as contributed by one or more of its units (Figs. 1, 2, 3, and 4).

Whenever multiple disabilities exist within one of these members, calculations must be made so that all of the factors can be interpreted in relation to the whole member without greatly exceeding 100 per cent.

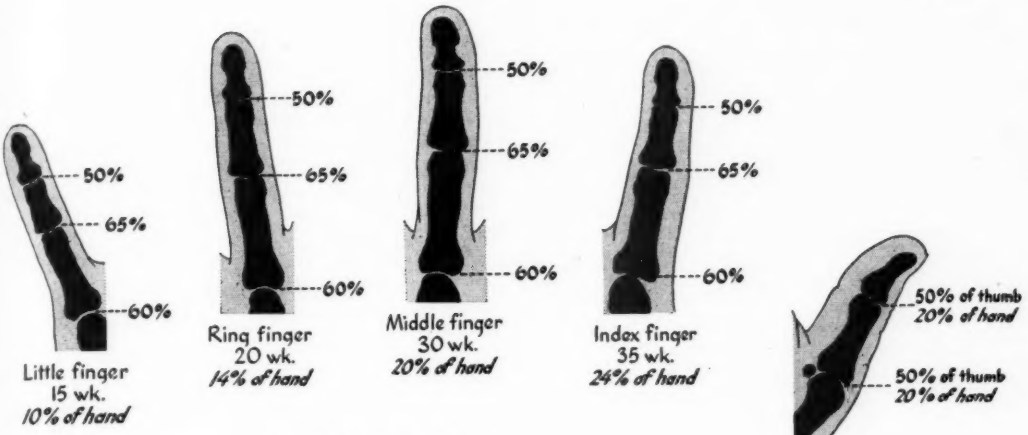
I shall endeavor to illustrate in these sketches the method by which disability is calculated. All of the details of my method of establishing these values cannot be completely explained in this brief discussion. More detailed explanations are now being prepared for a later publication.

In that the function of the phalanx is demonstrated through the motion in its corresponding joint, the disability of each phalanx can be calculated by simply determining the percentage of lost motion, out of a possible normal, using the opposite member for the normal standard.

If the distal phalanx of the thumb has lost 35 degrees motion out of a possible 70 degrees, the percentage of lost motion is 50 per cent (35/70 = 50%). A loss of 30 degrees motion in the proximal joint of the thumb out of a possible 60 degrees, likewise, constitutes 50 per cent loss of the proximal phalanx (30/60 = 50%).

The illustration in Figure 1 reveals that each of these joints has a value of 50 per cent of the thumb. Therefore, 50 per cent lost motion in each joint would reveal 50 per cent disability to the thumb on the basis of the following calculations:

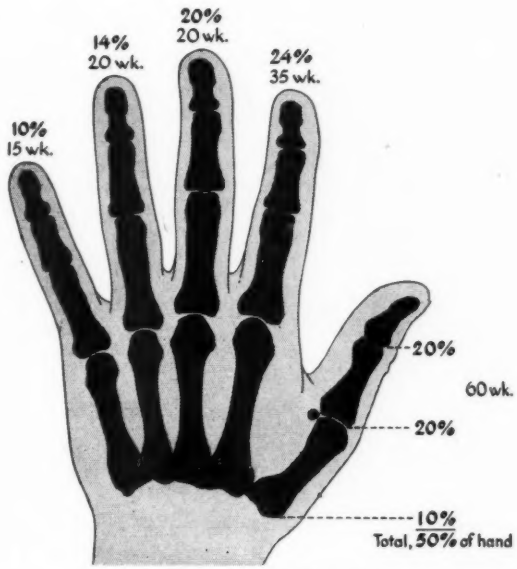
50% (loss to distal joint) × 50% (value of distal joint) + 50% (loss to proximal joint) × 50% (value of proximal joint) = 50% (disability to thumb).



Industrial value of fingers

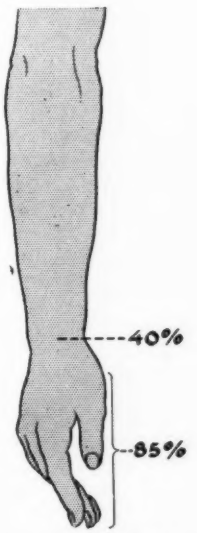
FORMULA: $\frac{50\%}{1} + \frac{65\%}{2} + \frac{60\%}{3} = 102.5\%$

Fig. 1.



Industrial value of hand
150 wk.
30% of "permanent total"

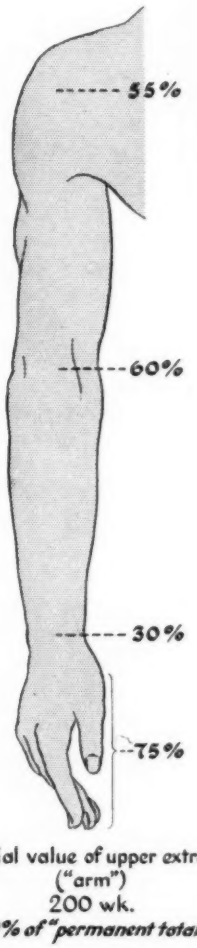
Fig. 2.



Industrial value of forearm
("hand and wrist")
175 wk.
35% of "permanent total"

Fig. 3.

Figure 1, illustrating the index finger, reveals that the distal phalanx has a value of 50 per cent of the finger; the mid-phalanx a value of 65 per cent of the finger, and the proximal phalanx a value of 60 per cent of the finger.



Industrial value of upper extremity
("arm")
200 wk.
40% of "permanent total"

Fig. 4.

The law specifies that the loss of or the loss of the use of a distal phalanx has a value of 50 per cent of the finger. The motion required in the middle and proximal joints to make the finger useful, can justifiably be given more value than the distal joints and, therefore, the value of 65 and 60 to the middle and proximal joints, respectively, can certainly be awarded for these.

These values would be awarded if each of these joints were involved alone, with rigidity. However, if more than one joint were involved with rigidity, their total value would exceed 100 per cent far in advance of complete uselessness. This is indicated in the following equation:

$$50\% + 65\% + 60\% = 175\%$$

It therefore becomes necessary to decrease the value of each additional joint, whenever more than the distal joint is involved, so that the final equation will not greatly exceed 100 per cent, when each of these three joints is completely rigid.

This equation must take into consideration the stipulation of the industrial law which specifies that the loss of use of the distal phalanx is equivalent to 50 per cent loss of the finger. The value of the distal joint accordingly cannot be diminished and, therefore, in the equation, its divisor is designated as 1.

The formula can be expressed as follows:

$$\frac{\%\text{ loss of distal phalanx} \times \text{value of distal phalanx}}{1} + \frac{\%\text{ loss of mid phalanx} \times \text{value of mid-phalanx}}{2} + \frac{\%\text{ loss of proximal phalanx} \times \%\text{ value of proximal phalanx}}{3} = \text{disability of the finger.}$$

If the numerical values for each of the joints of the finger, as illustrated in Figure 1, are substituted in this formula, it reveals the following:

$$\frac{50}{1} + \frac{65}{2} + \frac{60}{3} = 102.5$$

The 2.5 per cent excess makes it possible for the fingers to be 100 per cent disabled and still retain 3 or 4 degrees of motion in each of its joints. This seems not unreasonable for such a finger would still be industrially useless, even though attached to the hand.

If we were to assume 50 per cent of the lost motion in each of the joints of the finger, the following formula and percentage disability would be obtained:

$$\underset{\text{(disability to the finger)}}{\frac{50\%\text{ of }50\%}{1}} + \frac{50\%\text{ of }65\%}{2} + \frac{50\%\text{ of }60\%}{3} = 51\%$$

Figure 1 also reveals the middle, ring, and little finger with identical value for each joint as has been described for the index finger. Calculations of disability in these fingers is figured in the same manner.

The legal weekly indemnity for each of the fingers is illustrated to be: 60 weeks for the thumb, 35 weeks for the index, 30 weeks for the middle, 20 weeks for the ring, and 15 weeks for the little finger. The hand has a specified legal value of 150 weeks. From these figures the relative values of the fingers and thumb, in relation to the hand, can be calculated. These calculations reveal the following:

Thumb	$\frac{60}{150}$	= 40%
Index	$\frac{35}{150}$	= 24%
Middle	$\frac{30}{150}$	= 20%
Ring	$\frac{20}{150}$	= 14%
Little	$\frac{15}{150}$	= 10%
	Total	108%

The law specifies that the sum of the disabilities in the fingers and thumb cannot be awarded in excess of

that which is awarded for a hand. The additional 8 per cent over 100 per cent, as indicated in the total for all the fingers, suggests then, that the fingers need not be completely disabled in order that an award of 150 weeks be given.

The drawing (Fig. 2) illustrates that the thumb and fingers and their respective metacarpals constitute the entire hand.

The apponens actions of the thenar portion of the hand enclosing the metacarpal of the thumb, contributes additional value to the hand not included in that which is manifest by the thumb alone, as illustrated in Figure 1, where the thumb had a value of 40 per cent of the hand.

The thenar portion of the thumb adds 10 per cent to this, making a total of 50 per cent value of the hand for the thumb and its metacarpal.

The metacarpals of the fingers possess no independent value to the hand other than of supporting the fingers which in turn transmit their combined function into a hand. Whatever value the hand may possess, as far as the fingers and metacarpals are concerned, is manifest through the action of the fingers. It therefore seems reasonable, when making a calculation of disability in the hand, to base it upon the function in the fingers and thumb.

Therefore, in order to determine the disability in a hand, the disability in each finger is first calculated as described in the preceding paragraphs. This disability is then multiplied by the relative value of each finger in relation to the hand.

The disability contributed by the thumb and its metacarpal is obtained by calculating the percentage of lost motion in each of the joints of the thumb and its metacarpal. These values are multiplied by the relative values of each of these joints and the product is the factor which contributes to the disability of the hand.

If an example can be used assuming 50 per cent disability in each of the fingers and thumb, the following equations will reveal the disability to the hand:

	Disability		Value to Hand		Disability Contributed to the Hand
Thumb-Distal joint	50%	×	20%	=	10%
Proximal joint	50%	×	20%	=	10%
Carpa-Metacarpal joint	50%	×	10%	=	5%
Index finger	50%	×	24%	=	12%
Middle finger	50%	×	20%	=	10%
Ring finger	50%	×	14%	=	7%
Little finger	50%	×	10%	=	5%
Total	50%	×	118%	=	59%

The excess of 18 per cent in the total values, constituting the hand, would allow approximately 10 degrees motion in each joint of the fingers and thumb and still retain 100 per cent industrial disability. A hand with only 10 degrees motion in each of its joints would not constitute an industrially usable hand.

It seems justifiable to be generous to that extent in calculating a disability. Furthermore, it seems legally warranted to be generous to that extent, for, wherein the law specifies a total weekly indemnity for the loss of all of the fingers and the thumb of 160 weeks, it specifies only 150 weeks for a hand. The law further specifies 175 weeks for a "hand and wrist." A complete analysis of all the fingers and the thumb including their metacarpals would constitute, for practical purposes, the loss of use of a hand and wrist. Therefore, 118 per cent disability of 150 weeks would be equivalent to 177 weeks, which is only one-half week in excess of the indemnity of a hand and wrist. This seems to indicate that the values which I have chosen are accurate enough.

Figure 3 discloses the industrial member of "hand and wrist." A hand and forearm not including the elbow joint is perhaps anatomically more correct.

It has already been stated that a hand receives 150 weeks of indemnity in accordance with the legal stipulation of the industrial law. A hand and wrist is stipulated as 175 weeks.

In order that the hand might continue to receive its full indemnity, whether as a hand alone or in "hand and wrist" disability, its relative value to a hand and wrist must be adjusted accordingly. Therefore, it is found that the hand, in this instance, must be given a value of 85.7 per cent of a hand and wrist (150/175 = 85.7%). Leaving off the fraction, 85 per cent is used as the whole number.

This would allow only 15 per cent for the wrist. The wrist when involved alone probably possesses more value than that, and therefore, the wrist value has been placed at 35% of the hand and wrist.

In adding these two values, it is again seen that the total exceeds 100 per cent by too great a margin (85% + 35% = 120%).

It therefore becomes necessary when multiple disabilities are involved to use a formula, wherein the hand unit is divided by one, and the wrist unit divided by two. This would leave the following equation:

$$\frac{85\%}{1} + \frac{35\%}{2} = 102.5\%$$

The excess of 2.5 per cent would be sufficient to allow a slight amount of motion in the hand and wrist and still constitute 100 per cent disability. In figuring a disability of a "hand and wrist" one first figures the disability in the hand as explained in a previous paragraph, then figures the disability of the wrist, and applies these figures to the formula as previously illustrated.

As an example, if we could again assume 50 per cent disability of the hand and 50 per cent disability of the wrist, we would obtain the following equation:

$$\frac{50\% \times 85\%}{1} + \frac{50\% \times 35\%}{2} = 51.2\%$$

It is here revealed that we have been generous to the extent of 1.2 per cent.

Figure 4 illustrates the industrial member of an arm (upper extremity). In this instance it will be noted that the hand and wrist receive different values than they received when evaluated on the basis of a "hand and wrist."

I shall again point out that a hand must receive an indemnity of 150 weeks, whether on the basis of a hand alone, on the basis of a "hand and wrist," or on the basis of an entire upper extremity. In that an entire upper extremity receives an indemnity value of 200 weeks, the relative value of the hand in this instance would be 75 per cent (150/200 = 75%). Likewise a "hand

and wrist" which receives an indemnity value of 175 weeks must continue to receive 175 weeks even though the disability must be based on the entire upper extremity (175/200 = 87.5%). Therefore, in this instance, the wrist value must then be placed at 26 per cent.

Applying these two figures to the formula reveals the following:

$$\frac{75\%}{1} + \frac{26\%}{2} = 88\%$$

This represents one-half of 1 per cent in excess of the legal relative value (88% × 200 weeks = 176 weeks). This represents one week in excess of the 175 weeks of specified indemnity. This seems to be accurate enough and is as accurate as can be obtained without using fractions.

Figure 4 illustrates that the elbow receives a value of 60 per cent and the shoulder a value of 55 per cent. Using the formula, to calculate the disability in the upper extremity wherein all the joints are involved, the following is obtained:

$$\frac{75}{1} + \frac{26}{2} + \frac{60}{3} + \frac{55}{4} = 121.7\%$$

The 21 per cent excess would allow approximately 15 degrees of motion in every joint of the upper extremity and still constitute 100 per cent. An upper extremity with no more motion than that would be industrially useless.

The above figures and description show my method of calculating disability in the upper extremity. Similar figures and similar methods have been used for the lower extremity. More details with regard to the origin of these figures and the method of arriving at this method of calculating disabilities will be explained in a subsequent publication.

Discussion

DR. GEORGE DUNN: Doctor Rice has presented a very interesting paper. He has obviously given this matter a great deal of study.

In estimating permanent disability, one must remember that the estimate furnished by the doctor must harmonize with the existing laws of that particular state, otherwise the estimate becomes worthless.

Doctor Rice emphasized the importance in his general considerations of estimating disability largely on objective findings. This, in my opinion, is a very important consideration.

I enjoyed the paper very much and it will be very interesting after this paper has been published to study it carefully.

DR. R. E. HULTKRANS: I would like to make reference to two articles by Doctor Dunn about estimation of disabilities which appeared in MINNESOTA MEDICINE, and also one by Doctor Henry and Doctor Evans on "Interpretation of Disability in Minor Joints in Total Disabilities of the Extremities."

Doctor Rice has done an excellent job in calculating this so well. I think that any attempt to simplify this thing is extremely difficult, but I would like to call attention to the articles on estimation of disabilities previously published by members of this society, which will fall in line with what Doctor Rice has said tonight.

DR. EARL C. HENRIKSON: I am not sure that I understood whether the subject of your paper is on the estimation of joint disability alone. Should the calculation of permanent disability be restricted to the estimation of joint action only? If the estimation is on the joints alone the question comes up as to the additional disability due to sensory disturbances as well as other changes. I do not think you mentioned anything in your paper other than joint disability.

DR. RICE: I mentioned briefly only a few of the factors that had to be estimated. Loss of motion is the greatest factor in the cause of disability. I, therefore, dealt with only this one factor at this time. There are many other minor factors which contribute to a disability. These will be discussed later in a more detailed publication.

Many of those factors are pretty much of a guess. A certain number of them can be fairly well estimated and I think I have devised a method for some of them. If the radial nerve has been severed in the area above the elbow, all power of extension of the wrist and fingers have been lost, and the individual possesses no range of motion in the wrist or fingers. He would, therefore, retain 100 per cent disability of the hand and wrist. Other nerves transmit motor power to certain muscles, the loss of which would in turn produce loss of motion in the joints supplied by such muscles. These lost motions can be measured, and the disability thereby calculated.

Loss of sensation to an important tactile area such as the distal ends of the fingers constitutes from 10 to 15 per cent loss to the distal phalanx. If the distal phalanx has a value of 50 per cent of the finger, the loss to the finger would then be from 5 to 10 per cent of the finger.

Loss of sensation to skin over a small portion of the arm or forearm offers no industrial disability, and should therefore be worth no compensation award.

Loss of strength in co-ordination, instability, deformity, et cetera, has some value, but most of these are manifested by loss of the use of a member or a part of the member. I maintain that the useful range of motion should be used as a criterion. I do not ask an individual to force his range of motion. I merely encourage him to demonstrate his useful range of motion, using that as the criterion for my calculations. In so doing, I have taken into consideration most of the other factors such as weakness, pain, instability, inco-ordination, et cetera, and see no need to add additional values for these.

DR. R. F. MCGANDY: I am very much interested in seeing this published. I would like to read it more carefully.

Do you compare one member with the other in arriving at a disability?

DR. RICE: Yes, surely. All of the measurements are calculated against the opposite normal. In calculating specific disability, the opposite normal is used just as has been explained with the lantern slide illustrations.

DR. MCGANDY: In the wrist you mentioned flexion and extension only and didn't take radial and ulnar deviation into consideration.

DR. RICE: Ordinarily you need not. If the patient has lost all of a major motion, that is all you need take into consideration. Under such circumstances, minor motions would have no industrial value. To give them value when the major motion is completely lost would deprive the injured individual of disability due him.

If dorsi flexion, plantar flexion, ulnar deviation, and radial deviation were divided and each given equal values, their totals could not exceed 100 per cent. Then if each of these were found to have lost 50 per cent of their individual motions, the disability to the joint would still be 50 per cent. Most joints when developing a limitation of motion, develop it essentially equally in all of its various areas. It is, therefore, necessary only to calculate on the basis of one of the arcs of motion, i. e., dorsi-plantar flexion (the major arcs).

(Continued on Page 170)

Minnesota Academy of Medicine

Meeting of November 14, 1945

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, November 14, 1945. Dinner was served at 7 o'clock and the meeting was called to order by the President, Dr. A. G. Schulze, at 8:05.

There were forty-five members and six guests present.

Minutes of the October meeting were read and approved.

Dr. Erling Hansen read the following Memorial to Dr. Horace Newhart, and a motion was carried that this be spread on the minutes of the Academy and a copy sent to Dr. Newhart's family.

HORACE NEWHART
1872-1945

Dr. Newhart was born in New Ulm, Minnesota, on December 9, 1872. His preliminary education was received there and at Carleton College at Northfield, Minnesota. His Bachelor's degree was received from Dartmouth, where his scholarship was won him the Phi Beta Kappa key. From Hanover, he went to the University of Michigan for his medical course.

After a short time in practice near Albany, where he married Anne Hendrick, he returned to Minnesota and settled in Minneapolis for general practice in 1901.

Later, he decided to specialize in diseases of the eye, ear, nose and throat and spent several years in Vienna under Neumann, Hayek, Rutin, Alexander, and others, who became his close personal friends. Dr. and Mrs. Newhart were in Europe at the outbreak of World War I, and have returned there several times for visits. Several of the Viennese faculty have from time to time been guests in their home in Minneapolis.

Dr. Newhart was on the faculty of the University of Minnesota Medical School since 1912, and for several years before his retirement from active teaching in 1941 was director of the Division of Otolaryngology. His chief interest was always in otology.

Since the early '20's, Dr. Newhart has been a champion in the field of prevention of deafness and conservation of hearing, together with the problems of those who have become hard of hearing. His almost fanatical zeal in this field will long be remembered by those who came in contact with him.

This zeal bore fruit in his home community and in many distant places in the establishment of routine hearing tests for school children, the improvement in audiometers for testing hearing, and the development of better hearing aids. For many years he was chairman of the committee of the American Academy of Ophthalmology and Otolaryngology which dealt with conservation of hearing. He was co-author of the manual which the Academy issued on hearing tests only a few days before his death. He served the Academy as its President in 1925. He was made an active member of the Minnesota Academy of Medicine in 1931.

In addition to the activities already mentioned, Dr. Newhart was a member of his local and state medical societies and the American Medical Association, the Minneapolis Society for the Hard of Hearing, which he organized and of which he was president from 1925 to 1929; the American Society for the Hard of Hearing, president 1927-1928; American Laryngological, Rhinological and Otological Society; the American Otological Society, president 1939-1940; American College of Surgeons; American Acoustical Society; The Minnesota Academy of Ophthalmology and Otolaryngology. He belonged to Sigma Chi and Phi Rho Sigma fraternities and to the Lafayette, Minikahda and Campus Clubs. His hospital connections were with University, Northwestern, Abbott and St. Barnabas hospitals in Minneapolis and Glen Lake Sanatorium.

Dr. Newhart died at Northwestern Hospital on July 9, 1945, after a short illness. Funeral services were conducted Thursday, July 12 at Lakewood Cemetery Chapel with Dr. Donald J. Cowling, president of Carleton College, conducting the services. He is survived by his wife, Anne Hendrick Newhart; a son, Ellwood H.; a sister, Grace, and two grandchildren, Anne and Sally.

The Executive Committee and members present approved Dr. Thomas Robert's request that his name be transferred from the Active to the Senior Membership list.

Upon ballot, the following men were elected to membership:

St. Paul.—Dr. Wallace Ritchie and Dr. Joseph Borg; *Minneapolis.*—Dr. L. R. Boies, Dr. E. F. Robb, and Dr. H. B. Hannah.

The scientific program followed.

SEROLOGY AND OBSTETRICS

RAE T. LA VAKE, M.D.
Minneapolis, Minnesota

Dr. La Vake's paper appears in this issue, page 130.

Discussion

DR. LEE BARRY, Saint Paul: Karl Landsteiner, in 1900 and 1901, grouped human blood into four groups. Because of the cumbersome methods of blood transfusion, blood grouping had more of an academic than a practical interest until after 1911. Landsteiner and Wiener, in 1940, found that 85 per cent of human individuals contained a factor in their blood which was called the Rh factor. Levine, in 1941, established the role of the Rh factor as the cause of erythroblastosis foetalis.

Dr. La Vake, in his paper, states that the A, B (basis of blood grouping) and Rh antigens are more than agglutinogens. They at times may act as toxins. This is especially true when the fetus possesses one or more of these toxins or antigens and the mother does not. He is of the opinion that a toxin-antitoxin reaction between the mother and child due to difference in blood factors is the cause of erythroblastosis foetalis, pre-eclamptic toxemia, eclampsia and premature separation of the placenta.

Dr. La Vake, closing: This paper merely purports to lay before you certain conclusions to be drawn from a concomitant study of serology and obstetrics. If these conclusions are true, they should give direction to our therapy of pregnancy toxemia. The question has presented itself, that if we assume that the lesions of pregnancy toxemia are due to a fetal toxin, which stimulates the production of an antitoxin in the mothers, an antitoxin that may injure or kill the fetus, may it not reasonably be assumed, that, as the lesions of toxemia pregnancy may be permanent and life shortening for the mother, so the lesions of the antitoxin may be permanent for the fetus, even though by blood transfusions we may ostensibly clear up the visible signs of the injuries and save the immediate life of the child. Experiments are now under way that may aid in settling this question. As you all know, snake venom is merely a concentrated saliva. Eighty per cent of humans are secretors, having the hereditary ability to secrete their antigens in the saliva. This is apparently a phylogenetic carry-over. This may explan the sinister appearance and actions of many human bites. If the toxin antitoxin relationship between fetus and mother accounts for pregnancy toxemia and erythroblastosis and allied fetal injury, it would suggest itself that, could a rattlesnake be attached to its host as a child is attached to its mother, the snake could be injured and killed *in situ* by the administration of appropriate antitoxin to the host. The attempt is being made to so injure young rattlesnakes by direct injection of antivenom, to see if permanent injury results if the attack is protracted over a long span of time and to the point of death under conditions analogous to those obtaining in severe cases of erythroblastosis. The results of this work will be reported to you later. It is important because though our responsibility is clear in attempting to cure erythroblastosis by transfusion after delivery, it is not clear that we are warranted in subjecting a woman to the increased risk of cesarean section if permanent and inhuman results are likely to obtain in the fetus, even though the immediate life of the fetus can be saved by transfusion.

PREGNANCY COMPLICATED BY IDIOPATHIC THROMBOCYTOPENIC PURPURA AND SARCOID DISEASE OF THE SPLEEN; SPLENECTOMY AND SUBSEQUENT NORMAL DELIVERY

Case Report

MARTIN NORDLAND, M.D., R. S. YLVISAKER, M.D. (by invitation), PAUL LARSON, M.D. (by invitation), and ROBERT REIFF, M.D. (by invitation)
Minneapolis, Minnesota

Dr. Martin Nordland, Minneapolis: The occurrence of purpurahemorrhagica in association with pregnancy may be a coincidence of two unrelated conditions. When it occurs, it is a serious matter. The majority of patients die and the infants seldom survive. In this case the spleen was removed after the pregnancy had progressed for five months and the patient later went through a full term normal delivery. This case report is presented because it was thought it might be of interest to this group and because of its rarity we wished to place the report on record.

Too much praise cannot be given to the internist in the management of this case. The preparation of the patient and the choice of the time of the operation are of paramount importance in the successful removal of the spleen, under these circumstances. In order that the report may be presented to you in an orderly fashion, I shall ask Dr. Ylvisaker to give you the history, together with his comment. Dr. Reiff will discuss the hematology, and Dr. Larson will present the problems concerned with the obstetrical delivery. After they have finished, I would like to discuss the problems related to the splenectomy as a therapeutic measure.

Dr. R. S. Ylvisaker, Minneapolis (by invitation): The case presented tonight is that of a twenty-six year-old white housewife, (N. P.) who was first admitted to Northwestern Hospital October 28, 1943, and discharged November 10, 1943.

Present Complaint: (1) Multiple petechiae and achymoses; (2) profuse menstruation.

Present Illness: Her illness began about six weeks before admission when she first noted "red spots" over the lower extremities and abdomen. After three or four days, larger areas appeared over the ankles, followed by many such over the legs, hips and arms. Her menstruation two weeks prior to admission was very profuse and she felt faint if she tried to be on her feet. It was of normal duration. She struck her breast in a fall shortly before admission and a large ecchymotic area developed at the site of injury.

Family History: Both parents are living and well; divorced; there are no brothers or sisters and the family history is otherwise non-contributory.

Past History: She had a severe diphtheria at one year of age, complicated by an abscess which developed at the site of injection of antitoxin. There had been measles at the age of six or seven. A tuberculous chest lesion was said to have been found at the age of eight, for which she had been kept out of school for one year. She had been under observation at Lymanhurst Clinic, Minneapolis, from that time until 1942. The Clinic records revealed that an x-ray of the chest taken in 1926 at the age of nine, showed slight enlargement and calcification of the hilus glands in both lungs and clear lung fields. There was no Mantoux test done at that time, but in 1931, at the age of 13, the 1:1000 Mantoux was negative, the 1:100 was 1 plus positive. In 1942, 1-1000 Mantoux was again negative.

She had been hospitalized for two weeks at the age of ten for acute pyelitis with complete recovery. There had never been a recurrence. She had had jaundice for two or three weeks at the age of fourteen. There was no bleeding tendency then. She had been confined to bed, also at the age of fourteen, for a

supposed heart disorder for a period of six months, but was later told there was no heart disorder. She was hospitalized December 28, 1941, with first and second degree burns and aborted shortly after this, with no excessive bleeding. No history could be obtained of any symptoms suggesting any allergic disorder and there had been no ingestion of drugs. She has one healthy child, three years of age.

Physical examination on admission was normal except for the presence of numerous petechial hemorrhages scattered over the lower extremities and a smaller number over the upper extremities and trunk, as well as several old and recent ecchymoses over the right breast, both hips, both legs and both arms. The spleen at first could not be palpated, but on the third day it was felt just below the costal margin. The blood pressure was 120/78.

Laboratory Findings: Blood: hemoglobin 13.2 grams, red cells 4,590,000, white blood cells 7,600; differential: p.m.n.'s 67 per cent, lymphos. 23 per cent, monos. 8 per cent, eos. 1 per cent, baso. 1 per cent. Bleeding time 2½minutes, clotting time 5 minutes 20 seconds. Platelet count 60,000 per c.mm. Prothrombin time 6'/4'35". Later platelet counts, 108,000 and 112,000. On the eleventh day the hemoglobin was 16.8 grams, white count 10,900.

The patient was given two transfusions of 500 c.c. each of citrated blood. This apparently completely controlled the purpura and a menstrual period during her hospital stay was normal except for slightly increased flow the first day.

She was readmitted to the hospital October 26, 1944, discharged November 8, 1944. She was now five months pregnant. She stated that she had been well until a few days previously when the purpura had recurred. Examination again revealed numerous petechiae and ecchymoses over the entire body, but most numerous in the lower extremities. The spleen was palpable. There was a bloody crust in the right nostril.

Laboratory Data: Hemoglobin 12.0 grams, red cell count 4,380,000; white count 10,400; platelets 110,000; later 140,000, 180,000, and 250,000. Bleeding time 2 minutes 25 seconds, clotting time 4 minutes 50 seconds. Clot retraction began in 4½ to 5 hours and was still incomplete in 24 hours.

She was given five transfusions of citrated blood during this admission with incomplete control of her purpura.

Her third admission to the hospital occurred on November 12, 1944, four days after her previous discharge. There had again been exacerbation of the purpura over the entire body, in the conjunctivae, and over the dorsum of the tongue. She had noted frankly bloody urine three days previously. The spleen was palpable. The fundus of the uterus was at the level of the umbilicus. The Mantoux test was negative with 1:1000 tuberculin.

Laboratory Data: Hemoglobin 13.5 grams, red cells 5,250,000, white count 11,800; p.m.n.'s 78 per cent, lymphos. 17 per cent, monos. 4 per cent, eos. 1 per cent. Bleeding time 40 minutes, clotting time 3 minutes 20 seconds. Ivy bleeding time 54 minutes; platelet counts ranged between 80,000 and 230,000. Cuff test was positive. Prothrombin time 20'/15'. Clot retraction was incomplete in 24 hours. Sternal puncture was done by Dr. R. Reiff and he reported a marked megakaryocytic hyperplasia. He stated that smear of the peripheral blood showed less than 25,000 platelets and they were distinctly abnormal in appearance.

She was given a total of seven transfusions of 250 to 500 c.c. of citrated blood with incomplete control of the purpura. It was, therefore, deemed unwise to allow her to go to term without more drastic measures to control her bleeding tendency. Moreover, it was felt that all the diagnostic criteria of a true, idiopathic thrombocytopenic purpura had been fulfilled. Splenectomy was therefore advised and was done November 21, 1944, by Dr. Martin Nordland. During the operation it was noted that bleeding stopped as soon as ligation of the pedicle was completed.

Pathological examination of the removed spleen revealed the presence of sarcoid disease. Postoperative convalescence was uneventful except for the devlopment of a partial atelectasis of the lower lobe of the left lung which cleared without further difficulty. X-rays of the lungs and the bones of the hands revealed no evidence of sarcoid disease and no skin lesions were observed at any time. She was discharged on the fourteenth postoperative day in good condition. No further purpura was noted.

Her last admission to the hospital occurred March 23, 1945, at which time she was delivered spontaneously of a normal infant without incident. The puerperium was normal.

Further details of the hematological and pathological aspects of this case will be discussed by Dr. Robert Reiff, obstetrical aspects by Dr. Paul Larson, and the surgical aspects by Dr. Martin Nordland.

I shall not burden you with any lengthy discourse on this disease, but it might be of value to review, briefly, the main landmarks in the development of our present concept of idiopathic thrombocytopenic purpura.

Werlhof first described essential purpura in 1775. This was an acute case in a ten-year-old child which recovered spontaneously.

Donne, 1842, was supposed to have originally described platelets (globulins).

Denys was the first to show a reduction of circulating platelets in Werlhof's purpura in 1887.

Hayem discovered poor clot retraction in 1896.

Wright, 1910, demonstrated that platelets arose from megakaryocytes in the bone marrow.

Duke, 1911, showed that clotting time was normal, but that bleeding time was increased.

Frank, 1915, first enunciated the theory that thrombocytopenia was due to a megakaryocyte maturation defect.

Kaznalson, 1916, theorized that there was an increased platelet destruction presumably in the spleen, and suggested the first splenectomy which was done for thrombocytopenic purpura.

Marrow biopsy was first introduced by Seyforth, 1923, and was simplified by Arinkin in 1929.

Limarzi and Schleicher, 1940, summarized our present concept of the role of the megakaryocyte in thrombocytopenic purpura and described the bone marrow picure.

Schwartz, 1945, showed the presence of high eosinophile counts in the marrow in allergic purpuras and low counts in essential thrombocytopenic purpura and commented on the prognostic significance.

As far as the association of purpura and pregnancy is concerned, apparently some type of purpura was first observed in pregnancy by Arand in 1765, but it was not until a century later that Barnes reported what is commonly considered the first case of true purpura in pregnancy. From that time on, scattered cases were reported until, in 1943, Burnett and Klass could find only sixty-eight cases in the literature. Forty of these they considered doubtful cases, eighteen symptomatic and only four true cases of thrombocytopenic purpura. They added one of their own.

Finn, 1944, found five other cases which he considered true thrombocytopenic purpura and added three cases from the New York Hospital records. He found only four cases in which splenectomy was done during pregnancy for the purpura: (1) case of Palowe, 1940; (2) case of Bernstein, Newman and Hitzig, 1939; (3) case of Phythyon and Lartz, 1943; and (4) case reported in a letter in the *Journal of the American Medical Association*, written by Grommen, referring to a splenectomy done by Garlock.

A uniformly high mortality rate in both mothers and infants when splenectomy is not done, is reported by all writers—usually 55 per cent to 60 per cent. Rushmore, 1925, reported that of forty-four mothers, twenty-six died, and of forty-two babies, twenty-seven died, seven of the fifteen infants showing evidence of purpura.

As far as we know, the case being reported tonight is the fifth case to be reported in which splenectomy has been undertaken during pregnancy. Undoubtedly there have been other cases which have not been reported. We have been informed of a case in which operation was performed at the Minneapolis General Hospital at about the same time as this case and in which the mother later was delivered normally of a normal infant.

Features of the disease during pregnancy probably do not differ from those in the nonpregnant state. However, Evans and Perry, 1943, reported a case in which striking improvement occurred during pregnancy with a rise in platelets to 770,000, and a normal bleeding time. In this case, there was a reappearance of symptoms and a fall in platelets after delivery. They commented on the difference in incidence of the disease in males and females and suggested an endocrine etiology of the disease. In all likelihood their patient was sensitive to some estrogenic substance.

The relationship of purpura hemorrhagica to sarcoid disease of the spleen is not certain. In the light of cases of purpura which have been reported, secondary to cirrhosis and other liver diseases, tuberculosis of the spleen and other infections, our case should probably be classified as secondary to the sarcoidosis of the spleen. The only other case of purpura hemorrhagica which we have been able to find reported as secondary to sarcoid disease of the spleen, was by Schwartz, 1945, who stated that sarcoid disease of the spleen was present in one of his thirty cases.

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* * *

ROBERT H. REIFF, Ph.D., University of Minnesota (by invitation): The day preceding splenectomy, the significant hematologic findings were as follows: peripheral blood smears were essentially negative except for a severe thrombocytopenia—platelets numbered less than 20,000 per cubic millimeter. Sternal puncture revealed a moderate neutrophilic hyperplasia and an enormous increase in both the relative and absolute number of megakaryocytes. These cells numbered 1,822 in the terminal 18 square millimeters of the smears (normal—approximately 56). Megakaryocyte morphology was normal except for the absence of granular zoning characteristic of platelet formation in the adult cells. Very few platelets were found in the marrow smears. The bleeding time was 50 minutes, clotting and prothrombin times were normal, the clot did not retract, and the cuff test was markedly positive. The spleen was palpable but not remarkably enlarged.

Six days postoperatively, the platelet count had risen to 484,000; it ranged between 440,000 and 500,000 during the next four days. Three months later, the count was 464,000.

The spleen weighed 275 grams. The capsule was smooth, and the follicles were visible on the cut surface. Microscopically, many of the Malpighian bodies were replaced by tubercles composed of epithelioid cells and containing giant cells of the Langhans type. The lesions differed from those of tuberculosis only in the absence of necrosis in the central portions. The pulp showed no changes. The pattern was interpreted as typical of Boeck's sacroid.

Platelet counts, even in the most experienced hands, are often inaccurate and should always be checked by a study of the stained smear as well as the retractility of the clot. Bone marrow biopsy serves a manifold purpose. First, it excludes many of the causes of symptomatic or secondary purpura such as aplastic anemia, aleukemic leukemia and other lympoblastomas, extensive carcinomatosis, lipoid storage diseases, and others. Second, it affords a method of obtaining a quantitative as well as qualitative estimate of the megakaryocytes.

As J. H. Wright demonstrated in 1906, it is through the fragmentation of the cytoplasm of the megakaryocytes that the blood platelets are formed. Limarzi and Schleicher first called attention to the marked increase in the number of megakaryocytes in the bone marrow in essential thrombocytopenic purpura. In the cases which we have had an opportunity to study, the megakaryocytes ranged from eleven to sixty times the normal number prior to splenectomy. In each of these cases, removal of the spleen was followed by a prompt rise in the number of circulating platelets and concomitant disappearance of abnormal bleeding. Since the oldest case in our series was operated less than four years ago, information regarding late recurrences is not available.

In two cases, bone marrow biopsy revealed, in one, a marked decrease and, in the other, a complete absence of megakaryocytes. The spleen was removed in both cases because of progressively profuse hemorrhage in the presence of conservative therapy. Both continued to bleed postoperatively with a fatal outcome.

The presence of a palpable spleen suggests a complication; in this case, sarcoidosis. The literature is in fair agreement that the spleen is not enlarged in uncomplicated essential thrombocytopenic purpura. It is interesting to note, in connection with the case under discussion, that tuberculosis of the spleen has been found in rare instances of thrombocytopenic purpura.

The occurrence of thrombocytopenia in some newborn infants of mothers with purpura (Wintrobe), the marked decrease of marrow platelets in the presence of abundant megakaryocytes, and the usual absence of splenic pathology would appear to support Frank's original hyopthesis that the mechanism responsible for the thrombocytopenia is to be found in a failure of the megakaryocytes to form platelets, rather than in splenic sequestration and destruction.

* * *

Dr. Paul Larson, Minneapolis (by invitation): The essential points in diagnosis, treatment, etc., of the blood disease have been well covered by the hematologist, internist and surgeon. I shall only outline the prospects of the mother and child as well as describe the uneventful outcome of a successful case, in which sarcoid also was present.

To date, thirteen cases constitute the total number of reported cases of thrombocytopenic purpura complicating pregnancy. Of these, only three have had splenectomies during pregnancy, while three others had the same operation before gestation. One other had her splenectomy the seventh day postpartum because of severe postpartum hemorrhage. This last case is the only death in the group and, curiously enough, the only one in which severe postpartum bleeding occurred. Undoubtedly more cases would come into this category as the statements of Bernstein, Newman, Hitzig, Phythyon, Lartz, and Grossman bear out, but no reports are found.

Because of the rarity of this complication of pregnancy, little is known. There apparently exists no causal relationship between this type of purpura and pregnancy. It is a coincidental complication. It may occur at any parity, although those reported were all multipara except two. It may occur at any time during gestation. While it may be true that all cases of purpura will eventually die of hemorrhage, strangely enough, only one of the entire group reported developed a severe postpartum hemorrhage.

Outcome of the Baby.—Here a great variance of opinion exists, but from these reported cases in the literature the following points are evident:

1. Five infants died, one of which had proven thrombocytopenic purpura. The other four died of prematurity or atelectasis.
2. Seven normal children were born, and
3. One bled until the seventh day, then cleared.

It is evident that if complete remission of the process occurs after splenectomy, the newborn more likely will not be affected. It is believed that this purpura may be congenital but not necessarily inherited. It is difficult to draw any conclusions from such a small group of cases but it would appear that the newborn has a better chance if splenectomy has been done prior to the pregnancy or if the disease is in a chronic phase as pointed out in Finn's article.

Case Report.—Mrs. N. P., aged twenty-seven, grav. 3, para. 1, married five and one-half years. Her first pregnancy terminated at term uneventfully and resulted in the birth of a child in good health (1940) weighing 8 pounds 4 ounces. She had a miscarriage in 1941 at two months' gestation following a severe burn. In 1943 she developed petechiae in the arms and legs during which time she also developed a profuse menstrual period which was unusual for her. At this time a diagnosis of thrombocytopenic purpura had been made after adequate study. Her last menstrual period was June 6, 1944. There was a moderate amount of nausea the first trimester, with the pregnancy continued uneventfully until October 26, 1944, when she was admitted to Northwestern Hospital with a repeat episode of petechiae on the arms and legs. Several transfusions were given and she was discharged November 8, 1944. The patient returned again November 12, 1944, with a recurrence of the petechiae plus gross hematuria. After proper preparation, the patient had a splenectomy November 21, 1944, following which she recovered uneventfully. The pregnancy then continued without incident until labor was induced with castor oil and crude extract of ergot on March 24, 1945, at full term. Labor was five hours, normal, and no excessive bleeding followed. Delivery of a 7-pound female infant was spontaneous. The puerperium was normal.

The details of the blood studies during this time have been covered thoroughly by the internist.

Dr. Nordland: In the acute phases of purpura hemorrhagica, the natural tendency is toward improvement. This makes it difficult to evaluate any therapeutic measure, without due regard to the natural course of the disease, whether treatment is administered by conservative methods or by splenectomy. Because the course of purpura hemorrhagica is varied, caution must be exercised in becoming overenthusiastic for splenectomy. Evaluation of the effects of operation as compared

with the course of the disease in unoperated cases must be made only after the lapse of many years. There can be no question that splenectomy is a very valuable procedure in suitably selected cases. As was noted in this case, many surgeons have described total cessation of bleeding as soon as the splenic pedicle was tied. Surgical mortality should not exceed 5 per cent. Of these, no doubt, some would have died even if splenectomy had not been done. Removal of the spleen should not be done during an acute phase. Transfusions are extremely valuable preceding splenectomy. Frequent small blood transfusions of 100 to 300 c.c. given at intervals of two to three days have been considered of most value.

The indications for splenectomy may be enumerated as follows:

1. If bleeding is severe and fails to subside after repeated transfusions, splenectomy should be seriously considered. If there is any suggestion of improvement, however, operation should be deferred. Splenectomy is safer in the quiescent stage. In the first episode of bleeding, especially in children, it is more probable that bleeding will subside spontaneously rather than become more severe.
2. Operation should be given greater consideration in females than in males because of the possibility of a serious episode at menstruation or during pregnancy, as well as because recurrences seem to be more common in females than in males (4 to 3).
3. In chronic or recurrent cases, in addition to the above considerations, splenectomy is advisable if growth or development or the social or economic status of the patient is seriously impaired by recurrences, even if the hemorrhage is only moderate in character.

Splenectomy is contra-indicated in:

1. Cases of doubtful diagnosis,
2. Cases of symptomatic or secondary purpura such as those associated with acute infections or caused by some drug.

The removal of the spleen is not considered a particularly difficult operation. It is well to note, however, that because of its high location in the abdomen, the approach is occasionally difficult. This is particularly true in a woman well advanced in pregnancy. Anatomically, it is well to remember that the blood supply of the spleen is very abundant. The splenic artery, next to the renal artery, is the largest vessel supplying any abdominal viscera and is all out of proportion to the organ itself. The splenic artery, coming from the celiac axis, divides itself into a series of branching networks, finally allowing blood to percolate through the interstices of reticulo-endothelial tissue. Therefore, the spleen should be thought of as an organ made up of blood vessels and a fracture of this organ should be considered as a tear in one of the most vascular tissues in the body.

Discussion

DR. S. E. SWEITZER, Minneapolis: This case to me is interesting from the fact that this woman had sarcoid of the spleen. Sarcoid was first described by Boeck in 1889 as a cutaneous disease and later it was divided into several manifestations. Later, Schaumann called sarcoid lymphogranulomatosis maligna. It may involve the tonsils, bones, lungs, and nodes, and there are also skin manifestations.

Snapper and Pompeu in 1938 reported 13 cases. The lungs were involved in 8 cases, 5 had skeletal involvement, 5 had ocular involvement, the spleen was involved 7 times, 7 had cutaneous involvement, and 4 had liver involvement.

This patient, possibly on her symptoms, may have had primary sarcoid in the spleen; if so, she had a rare type of sarcoid. I do not remember seeing any individuals with sarcoid who had cutaneous hemorrhages.

The meeting adjourned.

J. A. LEPAK, M.D., *Secretary.*

SEROLOGY AND OBSTETRICS

(Continued from Page 132)

An understanding of the toxin-antitoxin relationship suggests the proper direction of studied therapy in the toxemias of pregnancy, erythroblastosis fetalis and in delayed transfusion toxemia.

Rh similar blood should be used in transfusion to prevent possible disaster if the woman has been isoimmunized by pregnancy or transfusion and to prevent isoimmunization that may interfere with her future ability to bear viable children.

CALCULATION OF DISABILITY IN THE EXTREMITIES

(Continued from Page 164)

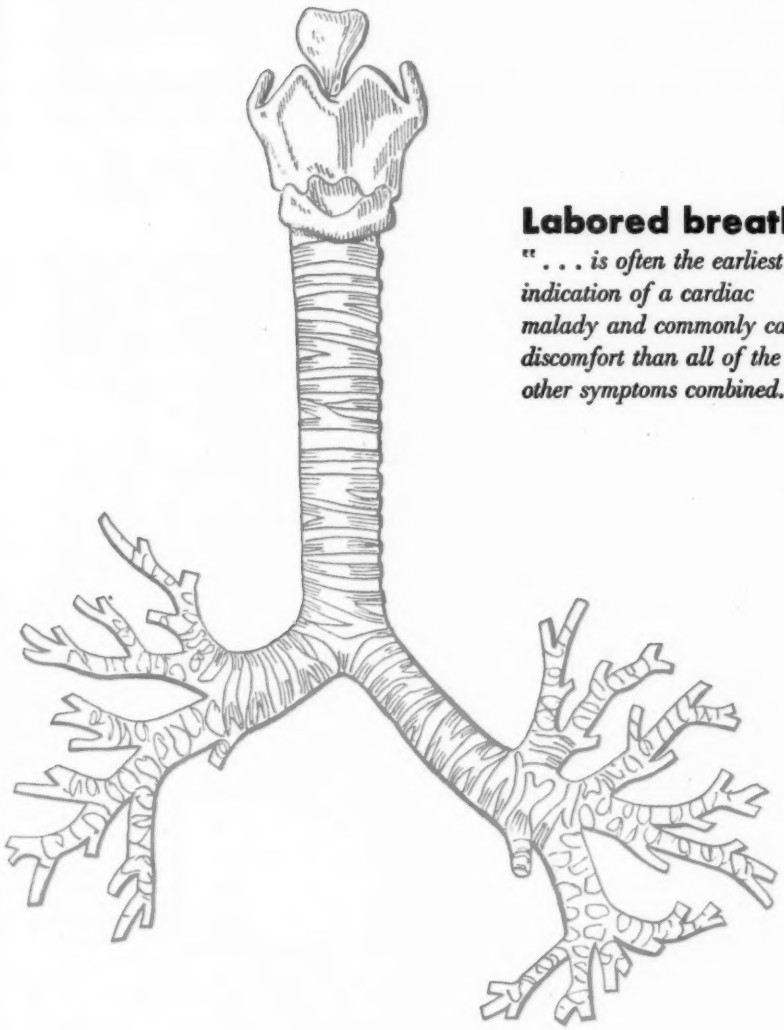
DR. MCGANDY: How do you figure pronation and supination in estimating a disability on a wrist?

DR. RICE: Pronation and supination of the forearm may come into disability estimation either in the elbow or in the wrist, depending upon from which it originates. If pronation and supination are completely lost, half of the wrist or elbow motion is lost. Dorsi-palmar flexion represents half of the motion in the wrist, and flexion-extension represents the other half of elbow motion.

DR. MCGANDY: I noticed that in your method of estimating a shoulder disability, you only mentioned forward elevation and backward elevation, saying nothing about internal and external rotation or lateral elevation.

DR. RICE: Ordinarily when one motion in the shoulder is lost, the other motions have a comparable loss. It would be extremely rare to retain all of forward-backward elevation, and lose only abduction-adduction. In normal activity, one does not use only one of the shoulder motions at a time. All three of the motions are being constantly and synchronously used.

ERNEST R. ANDERSON, M.D.
Recorder



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Reports and Announcements

MEDICAL BROADCAST FOR FEBRUARY

The following radio schedule of talks on medical and dental subjects by William O'Brien, M.D., Director of Postgraduate Medical Education, University of Minnesota, is sponsored by the Minnesota State Medical Association, the Minnesota State Dental Association, the Minnesota Hospital Association and the University of Minnesota School of the Air.

2—11:30 A.M. KUOM-KROC-KFAM	Medicine in the News
6—11:00 A.M. KUOM	Your Nervous System Directs your Life
7— 5:15 P.M. WCCO	He, Too, Is Ill
9—11:30 A.M. KUOM-KROC-KFAM	Medicine in the News
13—11:00 A.M. KUOM	As We Grow Older We Can Be Well
14— 5:15 P.M. WCCO	Give Him a Chance
16—11:30 A.M. KUOM-KROC-KFAM	Medicine in the News
20—11:00 A.M. KUOM	We Inherit Some Traits But We Acquire Others
21— 5:15 P.M. WCCO	The Job's the Thing
23—11:30 A.M. KUOM-KROC-KFAM	Medicine in the News
25— 4:45 P.M. WCCO	Your Community Hospital
27—11:00 A.M. KUOM	Stimulants and Narcotics Can Be Very Harmful
28— 5:15 P.M. WCCO	Teeth and Personality

ANNUAL PRIZE CONTEST

The American Association of Obstetricians, Gynecologists and Abdominal Surgeons Foundation announces that the annual prize contest will be conducted again this year.

For information address Dr. James R. Bloss, Secretary, 418 11th Street, Huntington 1, W. Va.

AMERICAN UROLOGICAL ASSOCIATION AWARD

The American Urological Association offers an annual award "not to exceed $500" for an essay (or essays) on the result of some specific clinical or laboratory research in Urology. The amount of the prize is based on the merits of the work presented, and if the Committee on Scientific Research deem none of the offerings worthy, no award will be made. Competitors shall be limited to residents in urology in recognized hospitals and to urologists who have been in such specific practice for not more than five years. All interested should write the Secretary, for full particulars.

The selected essay (or essays) will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Netherland Plaza, Cincinnati, Ohio, July 22-25, 1946.

Essays must be in the hands of the Secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis, Tennessee, on or before July 1, 1946.

NATIONAL GASTROENTEROLOGICAL ASSOCIATION AWARD CONTEST

The National Gastroenterological Association announces the establishment of an Annual Cash Prize Award of $100 and a Certificate of Merit for the best unpublished contribution on Gastroenterology or allied subjects. Certificates will also be awarded those physicians whose contributions are deemed worthy.

Contestants residing in the United States must be members of the American Medical Association. Those residing in foreign countries must be members of a similar organization in their own country. The winning contribution will be selected by a board of impartial judges and the award is to be made at the Annual Convention Banquet of the National Gastroenterological Association to be held at the Hotel Pennsylvania in New York City on Thursday evening, June 20, 1946.

All entries for the 1946 prize should be limited to 5,000 words, should be typewritten in English, prepared in manuscript form, submitted in five copies, accompanied by an entry letter, and must be received not later than May 1, 1946. Entries should be addressed to the National Gastroenterological Association, 1819 Broadway, New York 23, N. Y.

AMERICAN COLLEGE OF SURGEONS

The first of the series of ten sectional meetings sponsored by the American College of Surgeons was held at the Hotel Radisson in Minneapolis on January 28 and 29, with surgeons attending from Minnesota, Wisconsin, North and South Dakota, Iowa, Nebraska, Saskatchewan and Manitoba.

Lectures at the opening session were "Treatment of Infection by Chemotherapy and Antibiotics" and "Injuries to the Bile Ducts." During luncheon "Care of the Veteran" was the subject discussed, and dinner was followed by a forum on "Graduate Training in Surgery."

Dr. James M. Hayes, assistant professor of surgery at the University of Minnesota, who was in charge of arrangements, conducted a clinical conference at the January 29 session.

The meetings on both days opened at 8:30 A.M. with medical motion pictures.

MAYO PROFESSORSHIP IN PUBLIC HEALTH

The first permanently endowed professorship in the University of Minnesota was recently created as the Mayo Professorship in Public Health when the Board of Regents accepted endowment of that chair by the

(Continued on Page 174)

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(Continued from Page 172)

Mayo Properties Association. To pay the salary, which will not exceed $10,000, a gift of $150,000 was made to the University by the Mayo Properties Association, which specified that over and above the income from that sum the total will be made up from the income of the Mayo Foundation for Medical Education and Research.

The School of Public Health was created in the Medical School about two years ago when Dean Diehl recommended that the then department of preventive medicine and public health become a school within the medical school, like the school of nursing. Dr. Gaylord Anderson has been head of the school from the first, although during the war he has been on leave as head of the division of medical intelligence in the office of the surgeon general, war department.

The Mayo Properties Association is the organization formed by the late Drs. Charles H. and William J. Mayo and their associates, to hold and manage the assets of the Mayo Clinic. The Mayo Foundation for Medical Education and Research is an educational division of the University of Minnesota on the graduate level. It was first endowed with $2,000,000 in 1917, which sum was subsequently increased by $500,000 in 1934. It carries on a broad program of graduate teaching, maintains laboratories, and will now contribute to the new professorship.

The School of Public Health is one of six regional training centers in this country having as a principal function the training on the graduate level of doctors, engineers and nurses in public health matters to become specialists. For ten years past the school has had federal support of about $15,000 a year. Columbia, Harvard, Yale, Johns Hopkins, and Michigan are other institutions which have such schools.

Before the war there were from ten to fifteen doctors and from ten to fifteen engineers taking graduate work in public health. These programs were given up during the war but the public health nurses, numbering from 125 to 150, were carried through in the usual numbers. There is also a curriculum in public health education. Besides these programs the faculty gives instruction in personal and public health to undergraduates in many fields, including medicine, engineering, education, home economics, nursing and the like.

Minnesota was designated a regular public health training center after the state health officers of the north central group of states expressed a desire to have the federal government take that action.

FREEBORN COUNTY SOCIETY

The annual meeting of the Freeborn County Medical Society was held on January 18 at the Hotel Albert in Albert Lea. Dr. William L. Palmer, veteran Albert Lea physician, was guest of honor at the dinner which preceded the business meeting.

Dr. Leo L. Donovan, vice president during 1945, was elected successor to Dr. Brand A. Leopard, retiring president. Dr. J. Albert Schulz is the new vice president and Dr. Robert Swanson is treasurer. Dr. John Paul Person, of Alden, was re-elected secretary.

RED RIVER VALLEY SOCIETY

The annual dinner meeting and election of officers of the Red River Valley Medical Society was held at the Wayne Hotel in Crookston on December 27. Dr. Orlo K. Behr, of Crookston, is the new president, replacing Dr. John Cameron, of Erskine. Dr. William W. Johnstone, of Thief River Falls, was elected vice president, and Dr. Chester L. Oppegaard, of Crookston, was re-elected secretary-treasurer.

Dr. John F. Norman, of Crookston, addressed the meeting on "The Prepaid Medical Plan," and general discussion of the subject followed.

About twenty members and their wives attended.

WINONA COUNTY SOCIETY

Dr. John Tweedy was elected president of the Winona County Medical Society at the annual dinner meeting held at the Hotel Winona on January 7. Dr. Herbert Heise is the retiring president. Other officers chosen are: Dr. John S. Hamlon, St. Charles, vice president; Dr. Paul Heise, secretary and Dr. Leonard F. Johnston, treasurer.

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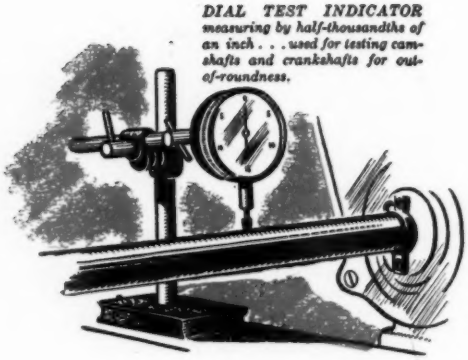

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Carlton, Minnesota

BLUE EARTH COUNTY

The Blue Earth County Auxiliary members donated to the party given by the Y.W.C.A. at their Club house, December 12, for the Young Mothers' Club and their children. Food and toys were given the children.

EAST CENTRAL

Each Central members elected new officers for the year when they met at the Cambridge Epileptic Colony. Mrs. Gonley was hostess. The new officers are Mrs. E. W. Miller, Anoka; Mrs. R. G. Swenson, North Branch; and Mrs. G. H. Tesch, Elk River.

GOODHUE COUNTY

The Goodhue County Medical Society were hosts to the Auxiliary members at the St. James Hotel, Red Wing, December 10. While the doctors conducted the business meeting, the ladies had a social evening.

HENNEPIN COUNTY

Mesdames H. E. Drill and Frederick Erb were in charge of the Silver Tea at the Medical Arts Building, Minneapolis, December 7. This tea is the annual philanthropic project for Sarahurst, the rehabilitation home for discharged tuberculosis patients. Carol Seagren inspired everyone with the Christmas spirit in the reading of a Christmas play, Julia Denecke, a flutist, added charm to the festive tea.

It's a thrilling record that the Hennepin County Auxiliary has achieved in the Bond booth. The E award has again been granted them; this time, when they had finished their work and exceeded the quota. Special tribute should be paid to the captain, Mrs. H. O. Altman, and to those members who gave unstintingly of their time and effort at the Medical Arts Building booth. Mrs. D. B. Mork points out that it is to their credit that they "stuck to their job and finished in a dignified fashion."

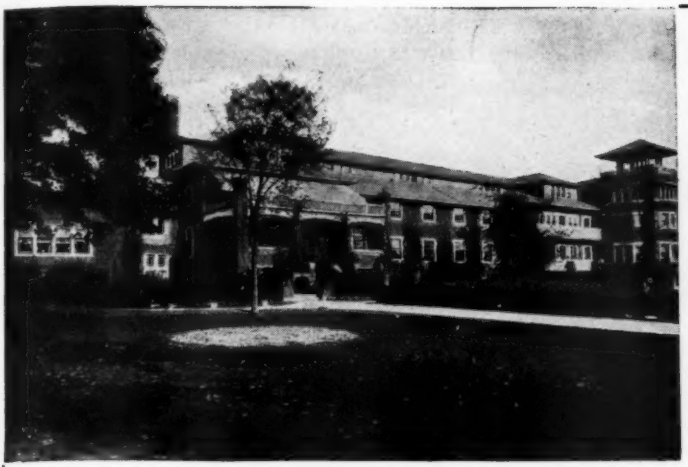
NICOLLET-LE SUEUR

A dinner meeting was followed by a business session at St. Peter on December 10.

RAMSEY COUNTY

One hundred fifty dollars was recently donated by the Ramsey County Auxiliary to the Red Cross Canteen conducted at the Union Depot in Saint Paul. Members continue to make cookies for the soldiers who pass through Saint Paul.

(Continued on Page 178)



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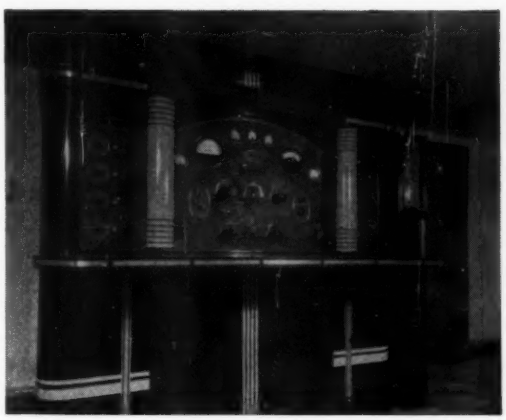

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(Continued from Page 176)

RENVILLE COUNTY

It is the custom of the Renville Medical Society to share their distinguished speakers with the Auxiliary members. The doctors arrange the dinner, holding it usually at the county seat, Olivia, and invite their wives. When there is no program, each group conducts separate meetings.

ST. LOUIS COUNTY

The St. Louis County Medical Society Members entertained their wives, December 13, at the annual county dinner meeting held at the Northland Country Club. After dinner, Walter Ridder, columnist, talked on his experiences during the war in the Scandinavian countries.

STEARNS-BENTON

In previous years, Stearns-Benton Auxiliary has given $5.00 to any student from the two counties who was among the ten winners in the state anti-tuberculosis essay contest. This year, since there were no winners from either Stearns or Benton counties, they recognized instead three students who received honorable mention. To stimulate interest in succeeding years, as well as to reward these students for their accomplishment, each was given $2.50 by the Auxiliary. Bernard Schaefer, junior at St. Boniface High School, Cold Spring; Helen Miner, Technical High School, St. Cloud; and Joan Reidar, ninth grade student, at St. Cloud Cathedral were the recipients this year.

WABASHA COUNTY

Wabasha County Auxiliary, just six members and these six members have twenty-three children! Yet they meet regularly and carry on informal discussions on weighty matters, such as *"Essentials for World Peace,"* their December subject. They place *Hygeia* subscriptions not only in the hospitals, offices and libraries, but also in the beauty parlors. They also make cancer dressings. On December 8, they met at the home of Mrs. A. G. Ashner at Wabasha.

WASHINGTON COUNTY

Mrs. J. W. Stuhr was hostess at Stillwater, December 9, for the group of which she is president. Christmas stories were read and gifts sent to two of their members who were sick.

Case finding among symptomatic patients cannot be expected to result in a majority of those patients being discovered at a minimal stage; and contact examination among associates of diagnosed patients, although yielding a high proportion of early cases, is limited in its scope, especially among urban residents. Mass roentgenography would appear to be needed if substantial reduction of cases which are advanced at the time of discovery is to be achieved.—Berwyn F. Mattison, M.D., *Amer. Jour. P. H.*, Nov., 1944.

In Memoriam

JOHN LeROY BURTON

Dr. J. L. Burton, the only physician at Buhl, Minnesota, was killed October 31, 1945, when his car went off the road.

Dr. Burton was born in Stewartville, Minnesota, February 5, 1904. He received his B.A. and M.B. degrees from the University of Minnesota in 1935 and his M.D. in 1936, interning at the Minneapolis General Hospital.

Dr. Burton acted as health officer and school physician at Buhl. He was a member of the St. Louis County Medical Society, the Minnesota State and American Medical Associations.

* * *

JOHN FRANCIS CURTIN

Dr. J. F. Curtin of Minneapolis was born at Lawrence, Massachusetts, October 17, 1889. He died December 25, 1945, at the age of fifty-six.

Dr. Curtin obtained a B.A. degree from Holy Cross in 1911, and an M.D. from Harvard in 1915. He interned at the Hartford Hospital and took postgraduate work at the Eye, Ear and Throat Hospital in New York City in 1919-20. During World War I he was instructor in ear, nose and throat surgery in the Army Medical School at Fort Oglethorpe, Georgia.

Dr. Curtin came to Minneapolis in April, 1920, and specialized in the treatment of ear, nose and throat diseases. He was a member of the Minneapolis Academy of Medicine, the Minnesota Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, the Hennepin County Medical Society, the Minnesota State and American Medical Associations. At the time of his death he was chief of the medical staff of Abbott Hospital.

* * *

ERNEST WESLEY RIMER

Dr. E. W. Rimer died of coronary thrombosis on December 22, 1945, at his home in Breckenridge, Minnesota. Dr. Rimer was born at Grand Junction, Iowa, June 20, 1882.

At the age of eighteen he joined the regular army and as a Sergeant in the 15th U. S. Cavalry saw service in the Philippines under Captain John J. Pershing before the close of the insurrectionary period. He also had the distinction of being the first white teacher on the island of Mindanao.

In 1906 he graduated from Valparaiso University with a degree in pharmacy and a B.A. degree. Two years later he received the M.D. degree from Rush Medical College in Chicago.

Dr. Rimer began practice in Breckenridge in 1912 but joined the colors again June 20, 1917. After training at the Army Medical College in Washington, D. C., he went overseas to England as First Lieutenant and became a Captain in the British Medical Corps. He served in France as surgeon of the Fourth Scottish

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Division and is said to have been for a time the only American surgeon in the front line trenches. He took part in the battles of Ypres, Cambrai, the Somme, Paschendale Ridge and Kimmel Hill. On one occasion he suffered from gas and at another time was reported killed. He recovered from a severe shell wound and after the Armistice was sent to Glasgow, Scotland, where he remained for eight months on duty in the Brigade Hospital. In 1919 he received the rank of Major in the British Medical Corps, and upon his return held the rank of Major in the U. S. Army Medical Reserve Corps. He received his discharge from the army at Camp Dix in 1919.

Dr. Rimer was a member of the staff of St. Francis Hospital, division surgeon for the Great Northern and Northern Pacific hospitals and county health officer. He was a member of the Park Region District and County Medical Society, the Minnesota State and American Medical Associations. He was a member and trustee of the local Baptist Church, a member of the Masonic Order, the Knights of Pythias, the Independent Order of Odd Fellows and the Elks. Many years ago he held the office of mayor, but all his outside interests yielded to his devotion to his profession.

Dr. Rimer is survived by his widow, and a daughter, Mrs. M. V. Young of Seattle, Washington.

* * *

JOHN CHANDLER SESSIONS

Dr. J. C. Sessions of Minneapolis was born at Atchison, Kansas, September 21, 1879, and died October 19, 1945, at the age of sixty-six.

He graduated from Central High School in Minneapolis and obtained the degrees of B.A. and M.D. from the University of Illinois in 1902. His internship was served in Cook County Hospital, Chicago, and the Minneapolis General Hospital. He took postgraduate study at the University of Illinois in 1908, and again in 1914.

During World War I Dr. Sessions served with the rank of Lieutenant-Colonel as division surgeon of the LaFayette Division.

He was a former member of the Hennepin County Medical Society, the Minnesota State and American Medical Associations.

He is survived by his widow, a son, Lieutenant John A. Sessions, and a daughter, Suzanne.

* * *

ARTHUR W. SHALEEN

Dr. A. W. Shaleen, a practitioner at Hallock, Minnesota, since 1903, died suddenly of a heart attack January 6, 1946, at his home.

Dr. Shaleen was born at Lindstrom, Minnesota, September 13, 1878. His father came to this country from Sweden in 1855 and served in the Sixth Minnesota Regiment in the Civil War, and for twelve years was a member of the State Legislature.

Dr. Shaleen attended Gustavus Adolphus Academy and College but left at the end of his junior year. He received his M.D. degree from the University of Minnesota Medical School in 1902, followed by an internship at the Swedish Hospital in Minneapolis. In 1910-11 he took postgraduate work in Vienna and Berlin and in 1918 served as Captain in the Medical Corps, being stationed at Atlanta, Georgia.

After World War I Dr. Shaleen resumed practice at Hallock, and it was due to his support that the Kittson War Veterans Memorial Hospital was established.

He was a member of the American College of Surgeons, a member of the Red River Valley Medical Society, the Minnesota State and American Medical Associations. He read and spoke Swedish, Norwegian, German, Italian, French, Spanish, Latin, and had considerable knowledge of Greek. He was an inveterate reader, and in his library were a number of volumes in French, Spanish and Italian. He donated 1,000 volumes to the public library at Hallock.

In 1930 Dr. Shaleen was a delegate from Minnesota to the Democratic National Convention in San Francisco. He was a Mason and a member of the Swedish Lutheran Church.

In December 16, 1937, Dr. Shaleen married Margaret Brighton Wallner who survives him.

* * *

FRANK DALE SMITH

Dr. F. D. Smith, since 1934 a practitioner at Rochester, Minnesota, died December 5, 1945, at the age of sixty-four.

Dr. Smith was born at Douglas, Minnesota, June 19, 1881. After attending Rochester High School, he graduated from the University of Minnesota Medical School in 1905.

He practiced twenty-four years at Kasson, Minnesota, and then moved to Rochester in 1934.

Dr. Smith married Pearl Olson of Oronoco June 12, 1912. His widow and two daughters, Dorothy E. Smith of Minneapolis and Mrs. Isabel J. Farnham of Rochester, and a granddaughter, Jane Page Farnham, survive him.

Dr. Smith was a member of the Presbyterian Church, a Mason, and a member of the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State and American Medical Associations.

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Of General Interest

The Board of Commissioners, Nobles County, has appointed Dr. John D. Waller, of Wilmont, county health officer.

* * *

Dr. James C. Crabtree, Saint Paul, and Dr. Alfred Kapsner, Starbuck, have established a partnership for the practice of medicine in Princeton.

* * *

Dr. James E. Ponterio, formerly of Shakopee, who recently returned from the South Pacific, has opened offices for general practice in West Saint Paul.

* * *

Ramsey County Board of Commissioners has announced the appointment of Dr. James H. McClanahan, of White Bear, as county health physician.

* * *

Dr. William A. Owens has terminated his practice in Montevideo and is now located in Minneapolis, where he is resident physician at the Deaconess Hospital.

* * *

Captain Marsh O. Perkins has opened an office with Dr. E. A. Smisek at 963 West Seventh Street, Saint Paul, after four years spent in service in France, Germany and Austria.

* * *

Lt. Colonel Arthur M. Mulligan, of Brainerd, who has been in military service in Hawaii, has been discharged and is now engaged in postgraduate work in Omaha, Nebraska.

* * *

Dr. Harry Bowers, Red Wing, has had word that his son, Dr. Robert N. Bowers, is on terminal leave after forty months of military service and will return shortly to his practice in Red Wing.

* * *

Dr. Louis A. Buie, of the Mayo Clinic, was elected a member of the Judicial Council of the American Medical Association at the meeting of the House of Delegates held early in December in Chicago.

* * *

Dr. H. Brauer, formerly of Sisseton, South Dakota, has been appointed health officer of Browns Valley, succeeding the late Dr. Bret V. Bates, who had been in office twenty years at the time of his death.

* * *

Dr. Ernest Mariette, superintendent of Glen Lake Sanatorium since it was first opened, was the guest of honor at the recent banquet which was given by the staff in celebration of the institution's thirtieth anniversary.

* * *

After three and a half years in the army, Dr. David Kaplan has returned to his practice in Saint Paul with offices in the Lowry Medical Arts Building. As Captain Kaplan, he was overseas from August, 1942, until his recent discharge.

Dr. Charles A. Aldrich, of the Mayo Clinic, was elected president of the American Board of Pediatrics at the annual meeting in Atlantic City. Dr. Aldrich had been secretary-treasurer of the Board since 1933, but had resigned just prior to the meeting.

* * *

Dr. Floyd A. Thompson, has resumed practice with offices at 245 South Snelling, Saint Paul. He attained the rank of Major and served three years in the South Pacific, having been stationed the last fifteen months with the 148th General Hospital on Saipan.

* * *

Major Eugene Muller, Saint Paul, has been discharged from the Army medical corps after four and a half years of service, twenty-seven months of which were spent with the 6th Army Division in the South Pacific. He will open an office in North Saint Paul.

* * *

Major Cecil A. Warren, Saint Paul, has been discharged from the Army where he served with the 121st Station Hospital. He spent twenty-six months in England and five months in France and Belgium. He is reopening his office at 373 Lowry Medical Arts Building, Saint Paul.

* * *

The War Department has awarded a Silver Star Medal to Captain William M. McConahey, Jr., who is now a Fellow in physical medicine at the Mayo Foundation. According to the citation the award was earned by "courageous action over and above the call of duty" on June 22 in the vicinity of Portbail, France.

* * *

Dr. Herbert Schmidt has been discharged from military service and has returned to his post at the Mayo Clinic. Duty on New Guinea was followed by hospitalization at Fitzsimons General Hospital in Denver. When Dr. Schmidt was again ready for assignment he was made a member of the staff.

* * *

After four years in military service, Dr. Bernard Street has returned to Northfield and entered into partnership with Drs. Joseph Moses and Warren E. Wilson. Dr. Street, a graduate of the University of Minnesota School of Medicine, was a major in the Army Medical Corps at the time of his discharge.

* * *

Dr. Ward Akester, who, since his release from the Army Medical Corps about eighteen months ago, has been with the Adams Clinic at Hibbing, has joined the staff of the Fergus Falls Hospital as assistant physician. Before entering service, Dr. Akester was in general practice in Marshall.

* * *

The St. Louis County Medical Society has announced the return to practice from military service of the following members: Dr. Charles M. Bagley, Dr. Arnold O. Swenson and Dr. Lawrence R. Gowan, all of Duluth;

(Continued on Page 184)

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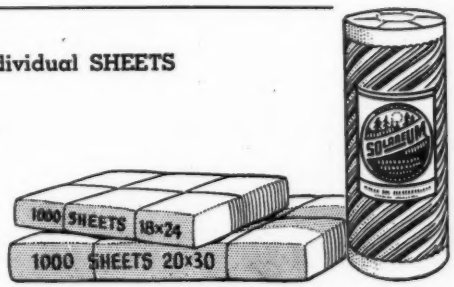
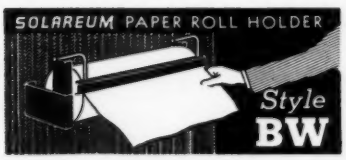


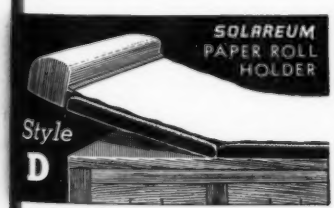
Style A Holder, for army metal and similar style tables without built-in headrest. For 18" roll, $4.25. With sliding parts for adjusting to 20" roll, $4.75. Nickel finish. Special finish, $3.50 additional.

Style B Holder, for Wood or Steel Examining Tables, to hold 20" roll, $4.75. For 24" roll, $7.25. Finished in White or Walnut. Special finish, $3.50 extra.

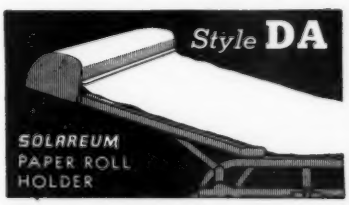
Style BT Holder, for wood or steel treatment tables. Takes 24" roll of SOLAREUM. Finished in white or walnut color, $7.25. Special finish, $3.50 additional.



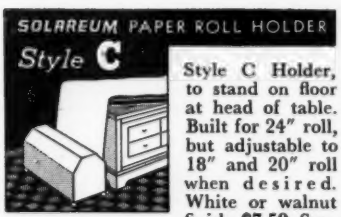
Style BW Holder, to fasten on wall instead of table, by 2 neat brackets. Secure and practical. for 20" roll, $7.75. For 24" roll, $7.90. White or walnut finish. Special finish, $3.50 extra.



tyle D Holder, for wood, steel-line, eel-tone and similar tables, to take 8" or 20" roll. Walnut, Mahogany, lack, Ivory or White finish, $8.50. wo-tone finish, $1.50 extra. Other pecial finishes, $3.50 additional.



Style DA Holder, for army, metal, and all similar tables, without built-in headrest. Holds 18" and 20" roll. Walnut, Ivory or White enamel finish, $8.50. Special finish to match your table, $3.50 extra. State whether for 18" or 20" roll.



Style C Holder, to stand on floor at head of table. Built for 24" roll, but adjustable to 18" and 20" roll when desired. White or walnut finish, $7.50. Special finish costs $3.50 more.

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MINNEAPOLIS MINNESOTA

(Continued from Page 182)

Dr. John D. Van Valkenburg, Floodwood, and Dr. Alvin Sach-Rowitz, Moose Lake.

* * *

Dr. Ernest R. Anderson was re-elected president of Asbury Hospital in Minneapolis at the annual staff meeting on January 3. Dr. Henry E. Hoffert was elected vice president and Dr. Harlan A. Alexander, secretary-treasurer. Drs. Chauncey A. McKinlay, Herbert M. N. Wynne, Thomas A. Peppard and Leonard K. Buzelle were appointed on the advisory board.

* * *

After three and a half years in the Navy, Dr. J. Allen Wilson has resumed the practice of internal medicine in Saint Paul. Dr. Wilson served eighteen months aboard an attack transport and took part in the invasions of Sicily, Salerno and Southern France. During his final year of overseas duty, he was senior medical officer with rank of captain.

* * *

Dr. Carl H. Coombs, discharged from the Navy in January, is again in practice at Cass Lake. During Dr. Coombs' absence, the care of the white population of the district was taken over by Dr. Zachariah E. House and Dr. Robert Campbell, both of whom had been looking forward to retirement for some time, but who instead carried on valiantly under a constantly increasing load.

* * *

Dr. E. J. Baldes, of the bio-physics section of the Mayo Cinic has been on a three months' trip in England, France and Germany for research in aviation medicine. Dr. Baldes traveled as a civilian for the Air Technical Command at Wright Field, Dayton, Ohio. On his return to this country the report of his findings was made directly to the office of the Air Surgeon in Washington, D. C.

* * *

Dr. B. A. Weis, Saint Paul, has been discharged from service in the Navy and has opened an office at 814 Lowry Medical Arts Building for the practice of internal medicine. Dr. Weis became a Commander in the Navy, was stationed eleven months at the Great Lakes Naval Hospital, spent two years in the Pacific, on land and sea, and spent the last four months at the Naval Hospital at Medford, Oregon.

* * *

Dr. Arden L. Abraham was made chief-of-staff of Miller Memorial Hospital, Duluth, at the annual meeting on January 4. Other officers elected at the same time are: Dr. Anthon J. Spang, vice chief of staff; Dr. Keith Fawcett, secretary-treasurer; and Drs. Herbert O. Hoff and Philip Eckman, executive committee. Dr. Mario Fisher was made executive committee member ex-officio.

* * *

Dr. Carl Krause, formerly of the Community Hospital in Fairmont, has returned to his home there, but will hereafter be associated with Drs. Herbert B. and Robert B. Bailey at the Bailey Hospital.

Dr. Krause has been in the armed forces since August, 1942. His first assignment was Fort Snelling, where

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he was in charge of obstetrics for the wives of servicemen for a year and a half. From there he was sent overseas and served with the 174th and 215th General Hospitals in England, France and Germany.

* * *

Dr. Olaf J. Hagen, who has been in practice at Moorhead and Fargo since 1907, was guest of honor at a testimonial dinner given by thirty-five friends and relatives at the Moorhead Country Club on January 15.

Dr. Hagen, now in his seventy-third year, is still active on the staff of St. Luke's Hospital in Moorhead. He is a former regent of the University of Minnesota and a member of both the American and the International Colleges of Surgeons, having been elected to the latter body at their meeting in Mexico City in 1941.

* * *

Two Mayo Clinic physicians, Dr. John S. Lundy and Dr. Eward B. Tuohy, were elected to offices in the American Society on Anesthesiology at the annual meeting in New York City in January. Elected president, Dr. Lundy took office immediately, while Dr. Tuohy was named president-elect for 1947.

Since 1946 is the one hundredth anniversary of the first public demonstration of the use of ether by William T. Morton in Boston, the year is expected to be of more than ordinary interest for the Society.

* * *

Dr. Robert Hollenhorst, of St. Cloud, has joined the Mayo Clinic, where he will be associated with the Department of Ophthalmology.

A graduate of the University of Minnesota School of Medicine, 1940, Dr. Hollenhorst served his internship at Abbott Hospital in Minneapolis and Ancker Hospital in Saint Paul. He was recently discharged from the Army with the rank of major after four and a half years in the Medical Battalion of the Sixth Infantry. Dr. Hollenhorst was on duty in New Guinea and the Philippines for more than two years.

* * *

Dr. Robert Wright, formerly of Jefferson, Wisconsin, is now with the Austin Clinic in Austin, where he is associated with Dr. David Anderson in the practice of general and gynecological surgery.

Dr. Wright was recently discharged from the air forces after two years in the Pacific Theatre, where he had served as a flight surgeon. He is a graduate of the Northwestern University School of Medicine, and following his internship spent a year in general surgery at the Ford Hospital in Detroit. He has also had special training in obstetrics and gynecology.

* * *

Dr. William O. Finkelnburg, recently discharged from the Army Medical Corps is now associated in practice with Dr. Irving W. Steiner in Winona.

Commissioned a first lieutenant in July, 1942, following the completion of his internship at Ancker Hospital, Saint Paul, Dr. Finkelnburg was sent to Hawaii, and later to the Solomons, New Caledonia and Okinawa. He was decorated with the Bronze Star for "meritorious service" in the Battle of Munda.

Dr. Finkelnburg is a native of Winona, where he

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resides with his parents, Mr. and Mrs. E. H. Finkelnburg.

* * *

Lt. Colonel Gerald H. Teasley, now a Fellow in urology at the Mayo Foundation, has been awarded the Legion of Merit for "performance of outstanding service." Formerly Director of the Military Personal Division, Office of Surgeon General, Washington, D. C., Dr. Teasley established a classification system for the Medical Department which was responsible for the best distribution of medical officers. He also prepared plans for demobilization to civilian occupations. According to the accompanying citation, Dr. Teasley's services "have been characterized by a high degree of proficiency surpassing that which might reasonably be expected of an officer in his position."

* * *

After an absence of five years in the Army Medical Corps, Dr. J. O. Meyer has returned to the Itasca Clinic at Grand Rapids.

A major in the General Headquarters Company, Seventh Division, Dr. Meyer participated in the initial landing on Attu and was on duty there and at Adack and Kiska until sent to Hawaii and assigned to the initial landings on Leyte. Dr. Meyer was at Okinawa from the assault until two weeks after V-J Day, when he was transferred to Korea.

While Dr. Meyer was away in service, Mrs. Meyer was employed at the University Hospital, where she was in charge of the Children's Clinic.

* * *

Dr. Charles Raadquist was made chief of staff of Hibbing General Hospital at the annual election of officers. Dr. Theodore A. Estrem was elected vice president and Dr. Arko, secretary. Department heads for the ensuing year are as follows: Dr. Clarence Jacobson, surgery; Dr. Carl N. Harris, medicine; Dr. L. William Morsman, opthalmology; Dr. Andrew Sinamark, otolaryngology; Dr. Edward W. Zeman, pediatrics; Dr. Theodore Estrem, obstetrics; Dr. A. Carlyle Tingdale, communicable diseases; Dr. Charles Raadquist, laboratory and x-ray. The resident physican is Dr. J. Cebula.

The annual banquet for the staff doctors is scheduled for the February meeting.

* * *

In an effort to remedy the sharp increase in communicable diseases reported by Dr. Frank J. Hill, city health commissioner, as a consequence of the housing situation in Minneapolis, Mayor Hubert H. Humphrey has organized a health advisory committee which includes the following from the medical profession: Dr. Charles E. Merkert, Hennepin County Medical Society; Dr. Frank J. Hill, city health commissioner; Dr. Donald W. Pollard, superintendent General Hospital; Dr. Edgar Huenekens, of the Board of Public Welfare; Dr. Gaylord Anderson, professor of public health, University of Minnesota.

In addition to assisting in the disposition of current problems, such as hospital expansion, construction of a new city hospital, modernization of General Hospital, policies of old-age assistance, care of convalescent and chronic cases, et cetera, the committee will function as a long-range planning board.

* * *

Absent from his offices in Canby for more than three years of service in the Navy, Dr. Leonard Monson returned to his practice shortly after the New Year. Later he will be joined by a partner, Dr. Raymond Chinn, with whom he worked in the South Pacific where they were attached to the same Marine Air Wing.

Dr. Chinn's home is in Washington, D. C., and he was in practice there when he was inducted into the Navy. Before moving to Canby, Dr. Chinn is taking some refresher courses.

Dr. Monson took part in the invasion of the Solomons with duty at Munda, New Georgia, Guadalcanal and Rendova. Returned to the States, he was assigned to recruiting service for a time, then was sent to Baltimore where he assisted in the commissioning of the *USS Typhoon*, a repair ship, and was aboard bound for Japan and nearing Hawaii when the war ended. Dr. Monson left the ship at Pearl Harbor and returned to this country. From then until discharged, he was at the Separation Center in Minneapolis.

* * *

Dr. Gordon L. Samuelson, who, prior to this entering military service had been practicing in Mankato for nine years, has re-opened his offices in the First National Bank Building.

Dr. Samuelson had been in service since September, 1940, when he enrolled in the Medical Field Service School at Carlisle, Pennsylvania. Following completion of his training, he was assigned to the adjutant general's office in Saint Paul and remained there until the National Guard was inducted into the regular army in January, 1941. He had twenty-six months of duty in the Aleutians and thirty-nine months overseas, with thirteen of them in the ETO in England, France, Belgium, Holland and Germany. He was a lieutenant colonel when discharged.

A graduate of the University of California, B.A., 1926, Dr. Samuelson received a B.S. from the University of Minnesota in 1929 and his medical degree in 1931. He has been a diplomate of the National Board of Medical Examiners since 1931.

During his terminal leave Dr. Samuelson took special work in urology at the University of Minnesota.

* * *

To meet the need of training more specialists in psychiatry and relocating medical men where psychiatrists are urgently needed, the National Committee for Mental Hygiene and the American Psychiatric Association have united to form a nationwide placement service. The service is designed especially to help physicians and psychiatrists make contact with training opportunities such as residencies, postgraduate courses and fellowships and to aid institutions in locating suitable candidates for appointment. Physicians interested in psychiatry are invited to send in full biographical statement including personal data, education, training, experience and special desires. Deans of medical schools, superintendents of hospitals and others employing or participating in the training of psychiatric personnel are

invited to submit full information regarding available positions and sourses. Foundations, universities and other agencies are asked to report pertinent fellowships in psychiatry, psychosomatic medicine, and child guidance. Inquiries should be addressed to Captain Forrest M. Harrison (MC) USN, National Committee for Mental Hygiene, 1790 Broadway, New York City 19, N. Y.

* * *

The Hennepin County Medical Society has announced the return to practice of the following members:

Dr. Clarence Arlander, 2300 Central Avenue, a commander in the Navy, was in the South Pacific for twenty-two months. He served in Navy hospitals in New Caledonia and the Russell Islands and was attached to the staff of Vice Admiral Aubrey Fitch for nine months.

Dr. Donald B. Frane, 1214 Forty-second Avenue North, a captain in the Army, was overseas for twenty-three months. He was with a signal construction battalion on the Ledo-Stillwell Road for seventeen months.

Dr. Erling T. Hauge, obstetrician, Medical Arts Building, a colonel in the Army Air Force, was on duty in the South Pacific for thirty-three months.

Dr. John A. Haugen, obstetrician, Medical Arts Building, a lieutenant commander in the Navy, was with the Marine Division in the South Pacific for twenty-six months.

Dr. Donald McKinnon has resumed his surgery practice in his offices in the Medical Arts Building. A commander in the Navy, he served twenty months in the South Pacific and at Sebo Naval Base, Japan. For outstanding service at Iwo Jima and Saipan he was awarded the Bronze Medal.

Dr. Alexander J. Ross, 2723 East Thirty-eighth Street, a captain in the Army, was in New Guinea for eleven months.

Dr. Donald S. Simonson, general practice, 4151 Lyndale Avenue South, served as a major in the Army in England and Fort Logan, Colorado.

Dr. James E. Trow, 5748 Nicollet Avenue, an Army captain, entered service in October, 1944. Following duty in Alaska and on the Alcan Highway, he was assigned to Ashford General Hospital, White Sulphur Springs, West Virginia.

Dr. Wilford F. Widen, 2929 Bloomington Avenue, a commander in the Navy, had nineteen months of duty in the Admiralty and Philippine Islands.

Dr. Robert L. Wilder, 1801 Lyndale Avenue South, pediatrician, served as a major in the Army. He was on duty in England and Fort Logan, Colorado.

Dr. William Wolf, general practice, 1417 West Lake Street, has been engaged in Veterans Administration work.

* * *

HOSPITAL NEWS

The new county hospital at Mora, Kanabec County was formally opened on December 26, and by the first of the year seven patients were listed. New Year's Eve brought an emergency case, and the first hospital baby was born. The total cost when all equipment has been installed will be $100,000. Capacity is twenty-

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two beds. The medical staff includes Drs. Willard F. Nordman, Clarence S. Bossert, and Alvin A. Peterson, of Mora; Dr. William T. Nygren, of Braham; Dr. Carleton G. Kelsey, of Hinckley, and Dr. Richard A. Whitney, of Cambridge.

* * *

The annual meetings of both Loretto and Union Hospitals in New Ulm were held on January 10, and the following officers were elected:

Staff president at Loretto Hospital, Dr. Joseph H. Vogel; vice president, Dr. Otto B. Fesenmaier, and secretary, Dr. Otto J. Seifert.

Dr. Fesenmaier was elected staff president at Union Hospital; Dr. Vogel, vice president, and Dr. Hjalmar E. Mortonbak, secretary.

* * *

Dr. Walter S. Broker, manager of the Wadena-Todd Sanatorium since it was opened in 1937, has resigned, effective December 1, 1945, and is now managing the Pleasant View Sanatorium in East St. Louis, Illinois.

Following his graduation from the University of Minnesota School of Medicine and his internship at St. Mary's Hospital in Minneapolis, Dr. Broker was in private practice for two years. In 1922 he entered the tuberculosis field at the sanatorium at Battle Lake and remained there until going to Wadena.

* * *

Dr. John Van Valkenburg was one of the speakers at the first annual meeting of the Floodwood Hospital Association held on December 17 to formulate plans for a membership drive and nominate a board of trustees.

Dr. Peter Ward, administrator of the Charles T. Miller Hospital and the Amherst Wilder Dispensary in Saint Paul, was elected president of the American Hospital Association at the meeting of the House of Delegates in Chicago.

* * *

The American College of Surgeons has published a directory of approved locations for surgical training chiefly for the use of returned medical officers. Approved locations for graduate training in 240 civilian hospitals in the United States and Canada, in thirty-two Naval, seven Veterans Administration and ten United States Public Health Service hospitals are listed. Nine locations in Minnesota are included. A copy of the directory may be obtained from the American College of Surgeons, 40 E. Erie Street, Chicago, Illinois.

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BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

MEN WITHOUT GUNS. By Dewitt MacKenzie. 152 pages, 137 illustrations. $5.00 Philadelphia: Blakiston, 1945.

This book contains a résumé of some medical aspects of World War II, together with a series of illustrations produced by the artists from their experiences in combat. The text, while not attempting to give a comprehensive account, gives some highlights of the work of the Medical Corps from the lay outlook. It rightly plays up the heroic record of the Medical Corpsmen. The text, however, merely offers a backdrop for the excellent portrayal of medical scenes by the artists. Many of these pictures have been seen in various publications printed by the Abbott Laboratories. As a collection of artistic and historical value this book is well worth while.

S. W. SHIMONEK, M.D.

DOCTORS AT WAR. Edited by Morris Fishbein, M.D., with the following contributors:

George B. Darling, M.D., Vice Chairman of the Division of Medical Science, National Research Council;
Harold S. Diehl, M.D., Member of the Directing Board, Procurement and Assignment Service, and Dean of the School of Medicine, University of Minnesota;
Major General David N. W. Grant, The Air Surgeon;
Charles M. Griffith, M.D., Medical Director, Veterans' Administration;
Major General Paul R. Hawley, Chief Surgeon, ETO;
Major General Norman T. Kirk, The Surgeon General, United States Army;
Major General F. Lull, Deputy Surgeon General;
Vice Admiral Ross T. McIntire, The Surgeon General, United States Navy;
Captain French R. Moore, Medcal Corps, United States Navy;
Thomas Parran, M.D., The Surgeon General, United States Public Health Service;
Brigadier General Fred W. Rankin, Chief Consulting Surgeon, United States Army;
G. Canby Robinson, M.D., LL.D., SC.D., National Director of the Red Cross Blood Donor Service;
Colonel Howard A. Rusk, Chief of the Convalescent Training Divison, Army Air Forces;
Brigadier General James Stevens Simmons, M.D., PH.D., D.P.H., Sc.D. (Hon.), Chief of the Preventive Medicine Service, United States Army. Illus. Price $5.00. New York: E. P. Dutton & Company, 1945.

This book is a brief survey of certain of the aspects of medical service in the war. It is a series of personal accounts by men concerned with some phases of the medical service. Each contribution has a self-explanatory title. Written by many, it necessarily is incomplete, and the style varies, but it is a readable and accurate account of the work of many agencies. Later, no doubt, more exhaustive treatises will be written.

GORDON C. MACRAE, M.D.

THE OSSEOUS SYSTEM. A Handbook of Roentgen Diagnosis. By Vincent W. Archer, M.D. 320 pages, 148 plates. Chicago: The Year Book Publishers, Inc., 1945.

This work is one of six Handbooks of Roentgen Diagnosis, written by different authors and discussing different aspects of roentgenology. In general, the author presents, briefly but very clearly and efficiently, the important abnormalities of bone tissue caused by trauma or disease.

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OBSTETRICS—Two-week Intensive Course starting March 11.

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principal headings: (1) Technique: Principles of Interpretation: Roentgen Anatomy; (2) Injuries to the Skeletal System Other than the Spine; (3) The Spine; (4) Bone Diseases and Abnormalities in Childhood; (5) Bone Diseases Occurring Principally in Adult Life. Under these main headings are given brief descriptions of various types of injuries and disease. The verbal discussions of individual disorders are adequately presented and accompanied by excellent illustrations. The author is careful in listing many points which may serve to avoid pitfalls or errors in diagnosis. Accordingly much emphasis is placed on proper technique of roentgenography in order to obtain sufficient knowledge of the part which is under study.

Although the book is intended for the occasional radiographer, it is considered to be useful also to those more experienced in the technical and diagnostic aspects of roentgenography of the osseous system.

R. J. Dittrich, M.D.

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